QUARTERLY

THE CHICAGO MEDICAL SCHOOL

VOLUME 5, NUMBER 2

JUNE, 1944

BUY WAR BONDS

QUARTERLY

Published Quarterly Under the Auspices of

THE CHICAGO MEDICAL SCHOOL

VOLUME 5, NUMBER 2 **JUNE, 1944**

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Editorials . . .

BONDS THAT BREAK BONDS

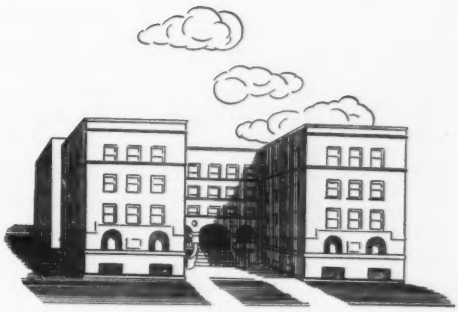
A NEW phase of the War has begun. Allied armies have breached the first walls of Hitler's "Festung Europa," and the puffed-up Third Reich is being deflated from three sides. But this is neither a time for rejoicing nor for complacency. Rather, it is a time for renewed determination to put every effort into helping our fighting men on towards victory.

For the task of liberating Europe and pushing the German armies back to their ultimate defeat will be no easy one. The enemy has been waiting for us to strike. He has strong forces with which he is striving to beat us back into the sea. And he is a desperate enemy, fighting a last-ditch stand. Before he is beaten, we must expect to hear of casualties beyond anything that we have experienced in this war. It is this knowledge that sobers us at this critical time, and that strengthens our resolve to back the attack in every way we can.

Our purchases of War Bonds and Stamps are assurances that our soldiers will continue to be supplied with the best of equipment. We have already seen the effects of our huge concentrations of planes, tanks and guns on the enemy fortress, but our material losses have been and will be great and must be replaced. The purchase of War Bonds to insure adequate replacements is the most direct and effective contribution we at home can make to the War.

The magnitude of the present military operations is almost inconceivable, but more than that, this undertaking constitutes the greatest

campaign in history to liberate conquered lands. It is reminiscent of the Crusades. Millions of lives will be affected by its outcome. But unlike the Crusades, the present struggle can have only one ending—complete liberation. It cannot fail. It shall not fail.



THE NEW STUDENT COUNCIL

THE value and need of a representative student organization in the School has long been recognized. The recently formed Student Council answers this need, and promises to become a highly useful and active body. The chairman and members of the Committee for a Student Council are to be commended for their part in making the Council a reality, for their initiative and efforts were largely responsible for its formation. (See Letter to the Editor.) It is now up to the student body to support and cooperate with the Council which they have elected. The Constitution of the Council wisely provides that meetings shall be open to the students. We urge the students to attend these meetings, take part in the discussions, and make the Student Council a vital and virile part of the School.

In this respect, it may also be pointed out that class officers are now in positions of greater responsibility than heretofore, because they automatically become members of the Council. The present members of the Council apparently are actively aware of their duties, and, with the cooperation of the student body, seem capable of making worth-while contributions to their fellow students and to the School. The maintenance of an active student organization, however, depends upon the seriousness with which the students elect their class officers in the future, for upon their efficiency and constructive efforts will rest the fate of the Student Council.

We of the QUARTERLY take this opportunity to extend our best wishes to the Student Council in its work ahead.

WATER METABOLISM

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I. THE DISTRIBUTION OF BODY WATER

The water of the animal organism is divided into compartments and separated from the external environment by membranes of varying permeability. The most impermeable membrane is the skin, which, from without inwards at least, bars the passage not only of large molecules like proteins and lipids but even of electrolytes, small organic molecules and water itself. From within outward, the skin does allow the escape of water vapor, and, of course, of sweat and sebum.

The membranes, if such they be, that form the boundaries of most tissue cells divide body water into two great compartments, that of intracellular and that of extracellular water. The former comprises about 50 per cent of the body weight, the latter about 20 per cent. Cellular activities are carried out by enzymes dissolved in intracellular water, and these activities are conditioned by a number of factors which must be maintained as constant as possible. Among these are the following:

1. The total osmotic pressure of the intracellular water.
2. The hydrogen ion concentration.
3. The absolute and relative quantities of the cations of the intracellular water.
4. The qualitative and quantitative colloidal content of the cells.
5. The influx of nutritive elements for cellular energy and repair.
6. The removal of waste products from the cell.
7. The temperature of the cell.

It is the function of the cell boundary and of the surrounding extracellular water to carry out the above functions. To permit this, the cell boundary must have a specialized permeability. It must be permeable to water and small organic molecules like glucose and urea. It must normally be impermeable to sodium and potassium, for the cells contain chiefly potassium and practically no sodium, whereas the principal cation of extracellular fluid is sodium, the potassium concentration being relatively low. There must also be some degree of impermeability to proteins, so that the proteins of intracellular and extracellular water shall remain in their respective compartments. On the other hand the cells must be permeable to hydrogen ions, carbonic acid and bicarbonate ions to allow free adjustment of the hydrogen ion concentration. This varying permeability of cell membranes is known to exist, but its mechanism is completely obscure. It is not absolute, varying somewhat in different tissues and in different species. There are also other limitations found in cell permeability, the reasons for which are not understood. There is, for example, an almost complete absence of chloride in most cells; also magnesium is in relatively high concentration in cells while calcium is relatively low.

Since it is now certain that there is little or no chloride in intracellular water (with the exception of that of red cells and probably of skin cells), it is possible to estimate the distribution of intracellular and extracellular water in any tissue or organ by analyzing the fat-free tissue for water and chloride and calculating the quantity of extracellular water by assuming that it contains all the chloride found and that its chloride concentration is that of other extracellular fluids (about 1.05 times the concentration of serum chloride). The quantity of water thus calculated, when subtracted from the total water analyzed, gives the intracellular water. Such a procedure can ,of course, be used only on the sacrificed animal. It has been utilized in brilliant studies by Darrow and Yannet and by Hastings and Eichelberger. In the intact animal or human subject, extracellular water has been estimated by injecting a known amount of a foreign substance which is believed to remain exclusively in the extracelluar compartment, and then ,after permitting thorough diffusion, determining the concentration of the injected substance in the serum which is regarded as representative of extracellular fluid. A correction must be made for the amount excreted during the period of the experiment. The substances used in this test have been thiocyanate and sucrose. In both types of methods, concordant results have been obtained in normal animals. However, the application of these methods to a study of the changed water distribution in disease begs the question in assuming that the cellular impermeability to chloride, thiocyanate, or sucrose continues unimpaired in the diseased state.

The extracellular fluid is the immediate environment of the living cells of the body. The higher mammals differ from their primitive marine forebearers in that the former "carry their ocean with

them." The success of the evolutionary process lay, among other things, in the development of the mechanisms for maintaining the constancy of this milieu, independently of the surrounding external environment, to paraphrase Claude Bernard. For, unless the extracellular fluid is constant, the intracellular fluid cannot be so, and life processes as we know them cannot long continue.

As has been stated, the extracellular water constitutes about 20 per cent of the body weight, or about 14 kg in a man of 70 kg. Of this 14 kg, about one fourth (or 3.5 kg.) is present as blood plasma and the remaining three fourths as interstitial fluids with which we shall include for convenience the lymph, cerebrospinal fluid, and the various secretions. The interstitial fluid is separated from the blood plasma by the capillary endothelium, and from the lymph by the lymphatic endothelium. Capillary endothelium constitutes a third type of membrane: it is permeable to water, electrolytes and crystalloidal organic compounds but not to lipids, proteins and substances which are normally adsorbed on proteins, like bilirubin. Interstitial fluid, then, is really an ultrafiltrate of plasma, just as if it were filtered through a collodian membrane.

Plasma contains about 7.3 gms. of protein per 100 cc., 4.5 as albumin, 2.5 as globulin, and 0.3 as fibrinogen. Interstitial fluid contains only a trace of protein, that which has accidently leaked through some unusually large endothelial pores, or that which has been formed in the cells and is on its way to the blood. Since the capillary endothelium is impermeable to protein, there is present in each capillary an osmotic machine which is capable, if unopposed, of drawing all the surrounding interstitial water into the capillary lumen. This colloidal osmotic pressure, normally equal to about 23 mm. of Hg, is however opposed by the hydrostatic pressure of the blood in the capillary, transmitted from the action of the heart. At the arterial end, this hydrostatic pressure has a value of about 32 mm. of Hg; at the venous end about 12 mm. Thus at the arterial end, the hydrostatic pressure, being greater than the colloidal osmotic pressure, forces fluid to filter into the interstitial space, while at the venous end, the higher colloidal osmotic pressure brings about a return of interstitial fluid into the capillary. It is the balance between these two opposing tendencies, aided by the simultaneous removal of interstitial fluid by the lymphatic system and of vascular fluid by the kidneys, as well as by the elastic resistance of the tissues, that keeps the volume of blood and interstitial fluid constant. There are, however, many other factors concerned with the homeostatic mechanism some of which will be discussed later. A disturbance in the relationship between colloidal osmotic pressure and the capillary hydrostatic pressure may produce edema or some of the manifestations of shock.

Edema is of three types. The so-called nephrotic type is produced by a lowering of serum protein concentration due to protracted severe albuminuria or to low protein intake. If the serum colloidal osmotic pressure drops appreciably below the normal 23 mm. there is an increased flow from the capillaries at the arterial end and a diminished return at the venous end. Thus there will be a lag in the return of fluid from the interstitial spaces, and if the amount of fluid accumulating is sufficient to be detected clinically, the phenomenon is called edema. This condition is seen most frequently in lipoid nephrosis, the nephrotic stage of glomerulonephritis, and in amyloid nephrosis. It has also been seen in protein starvation, as in the German prison camps during the last war.

A second type of edema, often called the nephritic type, is produced by damage to the capillary endothelium allowing the passage of protein into the interstitial fluid. If the protein content of this fluid should rise, say, to 2 per cent, then the effective colloidal osmotic pressure would not be due to 7 per cent protein solution as normally, but to a concentration less than 7 minus 2, or less than 5 per cent. This lowered colloidal osmotic pressure will allow greater filtration at the arterial end of the capillary and less resorption at the venous end. Such edema is seen in generalized form in acute nephritis, is seen locally in blisters, burns, inflammation, and frostbite.

The third type of edema is sometimes called cardiac edema. It is produced by increased venous pressure. If the hydrostatic pressure at the venous end rises, say from 12 to 20 mm., there will be less tendency for fluid to return at the venous end. This type of edema is seen in cardiac decompensation, in the use of the tourniquet, and in varicose veins. It is probably always complicated by some nephritic type of edema because of damage to the endothelium by anoxemia and CO_2 accumulation.

Still another kind of edema, lymphedema, as dramatically demonstrated in elephantiasis from filaria infection, is probably a special case of nephritic type of edema. Here obstruction of the lymphatics prevents the removal of the small amount of protein

constantly leaking into the interstitial fluid, so that eventually a concentration of interstitial protein is reached equal to that seen in the nephritic type of edema. Myxedema may also be a special case of the nephritic type of edema.

The function of the lymph system and the mechanism of the flow of lymph has remained a controversial subject. The most reasonable theory appears to be that of Peters as expressed in his book Body Water (C. C. Thomas, Baltimore, 1935). Lymph vessels serve to remove protein, lipids, and particulate matter from the interstitial spaces. They form a closed system like blood vessels, yet proteins and other large particles are able to enter into the lumina of the lymph vessels, apparently not by diffusion but by special phagocytic activities of the lymphatic endothelial cells. (If this be true, these cells resemble in this respect the reticulo-endothelial cells). Once in the lymphatic the protein does not diffuse back but accumulates to a concentration that exerts enough colloidal osmotic pressure to suck interstitial fluid into the vessel. This initiates the movement of the lymph and the flow is sustained by muscular movement and by the presence of lymphatic valves. Particulate matter is filtered out at the lymph nodes, but protein is returned to the blood circulation via the thoracic and other ducts.

The role of water metabolism in shock can be no more than mentioned in this lecture. The primary shock of severe hemorrhage or of extensive burns or of violent trauma is associated with a drop in blood pressure and a marked diminution in blood volume. In hemorrhage this reduction of blood volume is undoubtedly primarily due to blood loss. In burns, it is produced by loss of serum at the site of the burn. But no such explanation can account for a similar diminution in blood volume in traumatic shock. Here it is probably due to accumulation of non-circulating blood in dilated stagnant capillaries. In any case, the blood volume does not automatically become restored, as in mild hemorrhage, by influx of interstitial fluid into the vascular system. If saline solution is administered intravenously, the blood volume is only temporarily restored, for in the presence of the damaged capillaries there is a rapid loss of this injected fluid into the tissue spaces, carrying with it serum protein and further depleting the blood store of protein. Only if the colloidal osmotic pressure of the plasma is restored by transfusion of blood, plasma, or plasma substitutes, can the blood volume be restored to and maintained at the normal level.

II. WATER BALANCE.

Under normal circumstances, the animal organism has no difficulty remaining in water balance. Water is consumed in response to thirst, which normally is manifested by a sense of dryness of the mouth and pharynx. This sensation is produced when the water content of the body is less than normal, provided there has been no great loss of salt. It also occurs when the salt content of the body is greater than normal, even without a loss of water.

It should be recognized that the drinking of fluids is only one of the sources of body water. The other two are the water of solid foods and the water of oxidation of ingested or endogenous organic substances. The following pattern for an average man on a 2400 cal. diet in temporate climate gives some idea of the magnitude of these three sources:

Water and other beverages	1200 cc.
Water of solid foods (about 70%)	1500 cc.
Water of oxidation (about 12 cc. per 100 cal.)	300 cc.
Total	3000 cc.

The body loses water by excretion through the following channels: the kidneys, the alimentary tract, the skin, the sweat, and the lungs. Under average conditions the sum of these outputs will balance the intake as follows:

Urine	1500 cc.
Feces	200 cc.
Skin (as water vapor)	700 cc.
Sweat	300 cc.
Lungs (as water vapor)	300 cc.
Total	3000 cc.

The loss of water vapor through the lungs and skin is called the insensible perspiration. The energy required for this vaporization accounts for about 24 per cent of the heat produced by the entire body. Since, according to Du Bois, this percentage is fairly constant under a variety of conditions, the insensible perspiration may be used as a measure of total energy output. However, the external environment has a pronounced effect on this loss, it being greater at low humidities and high temperatures.

Sweat is secreted when it is necessary to use additional methods to cool the body. It is produced during increased energy output even in normal external environment and without increased energy output if the external temperature rises to 30° C or

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THE TECHNIQUE OF PSYCHOTHERAPY—II

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THE goal of psychotherapy as that of any medical treatment is to cure the patient of his ailment. In psycho-neuroses that cannot be accomplished without a change in the patient's attitude to his present life problems which he evades by being sick. The present attitude of the patient results from the impact of his personal life style and the acute life situation for which he is not prepared. As it is generally impossible to alter the objective life situation, therefore it is necessary to change the patient's whole life plan so that he can meet more successfully the tasks of life which confront him.

Uncovering what is taking place in the patient, revealing the purpose which dominates his present behavior, is part of an organized effort to challenge the patient to a new orientation.

However, words have their limitations. Explanation is only one method of education. No education can be effective if it remains totally on the verbal level. Presentation of ideas and rationalization must be supplemented by vital experiences, by psychological action. Every psychotherapy reaches a point where the patient, for the time being, is not amenable to logic. The treatment must become dramatic to jolt the patient out of the complacent security which he provided for himself by using his illness as a protection from life.

Every therapist develops his own technique to overcome such a deadlock and psychological blockage and to create experience which make the patient receptive to further rational discussion. A change in the thought process of the patient is still the fundamental factor in psychotherapy; for upon understanding alone rests the change of the life plan, and hence, the success of the therapy. For this reason we must warn against, not only any over-estimation of psychological tactics, but also, above all, against a careless and too liberal application of them. They may easily endanger the whole treatment. Indeed, they put the heaviest demand upon the therapist's sense of tact. For it may happen only too easily that the patient will feel duped. If this is the case, further cooperation between therapist and patient is definitely disturbed. Moreover, the use of manoeuvres may cloud the atmosphere of complete confidence which is so necessary. Finally, the position of the therapist is jeopardized by the use of cunning because he then appears to the patient in a position of especial superiority, a thing which from the viewpoint of psychotherapy leads to a state where the patient finds it easy to make the therapist responsible for his own failure. The failure, then, is not due to lack of readiness on the part of the patient, but rather to the inability or the errors of the therapist, who is more likely to be exposed to this criticism once he has entered the field of influencing through management. Nevertheless, sometimes artifice cannot be avoided when other ways of appealing to the patient's intelligence seem impossible for the time being.

Naturally I cannot enumerate all the devices which we have acquired. I shall merely attempt to point out some of them. How the individual therapist applies these principles is, of course, entirely dependent upon his creative faculties and his ability to put himself in his patient's place.

First we might mention the tactic of surprise, of startling . We must remember that when the therapeutic work comes to a standstill, this stagnation is caused by the fact that the patient has secured himself completely in the maintenance of his accustomed life-style, even against the psychiatrist—and these safeguards cannot be broken through by words or simple information. Astonishment, first of all, makes the patient listen; he does not know what he is up against. Thus, he will have already given up his set attitude and become receptive to any remarks which may help him in seeing his present situation more clearly. Naturally this surprise must not have in it anything that might hurt the patient's feelings or set him against us. We must merely do the thing that he would least expect. Often it is enough if we abandon an opinion which we have held previously or express the opposite of what, with justification, he might expect of us. He is then forced to get new bearings, to work out new devices for obtaining his old stability. Often through discussion of these problems the contact is reestablished. The element of surprise is actually in a measure fundamental to all therapeutic stratagems.

It is the same when we suddenly appropriate the patient's point of view and, contrary to our usual practice, attempt to defend this standpoint of his with his own arguments. In the moment of surprise the patient often suddenly takes over the atti-

tude of his opponent, in our case that of the therapist. He proves in this way that he had not really believed in his own former opinion and that he had acted rather in opposition to common sense, to the play-rules of human society, and also contrary to all logic. That which hitherto seemed incontrovertible to him contradicts itself when no one else contradicts it. Let anyone else seriously attempt to defend his obviously illogical attitude and the error becomes immediately apparent to the patient. To carry on such a discussion with changed roles, a high degree of diplomacy is necessary, of course, so that the patient will not feel ridiculed from the start and reject this procedure. However, when it is possible to set such a discussion in motion, even occasional exaggeration will do no harm.

A young girl of nineteen who suffered from states of fear suddenly felt very ill in the course of treatment. She was obsessed by the idea that she had a stomach cancer. She felt that she had to stay in bed, and plagued her parents, who were obliged to quiet her continually. I tried to demonstrate to her that by this behavior she was endeavoring to evade the approaching first problems of her love-life and that, being a very pampered child, she wanted to do what she always had done in difficult situations—force her parents to give her special attention and sympathy. It was of no avail; she did not understand anything and kept on complaining. It was impossible to draw this otherwise very intelligent girl into a reasonable discussion. At the same time she refused to eat and actually lost weight at an alarming rate. Then all at once I remarked to her that perhaps she really did have a cancer. In a flash the situation changed. First she was surprised, then she objected that no physician, though her parents had already consulted many, had found any signs of cancer. Besides, she was much too young for cancer, etc. All that those around her had unsuccessfully sought to prove to her before, she now used against me. I insisted, however, that it might be a cancer. Then she began to weep and was—as I found out later—justifiably angry with me, especially since I had recommended that no one should contradict her. But the thought of cancer had definitely been abandoned, and the path had again become open for further treatment.

Thus we come to one of the most dangerous weapons at the command of the psychotherapist and one which all of them occasionally use: this is irony. An ill-advised dose of it can result in the patient's staying away forever. But, discreetly used, nothing can illuminate a situation in which the patient tries to evade the logic of our arguments so well as an ironical remark. The patient must first consider how such a remark is intended, must, as it were, work out for himself the therapist's trend of thought. And, what is even more important, something is made clear to him without his really being able to contradict. Moreover, the point of the argument, the solution which had not been possible to present before, through discussion, is suddenly stripped of its seriousness and turned into the comic. The therapist must not forget, however, the fact that nothing has quite so wounding an effect as irony. If wounds are not to be inflicted, irony must be set forth in an atmosphere of benevolent consideration and every reaction of the patient carefully sounded. If this is done, one sentence may often explain more than might be possible in a long debate.

Irony is also an ingredient somehow of all finesses, a reason why the latter are so dangerous. Now this irony may be concealed in various ways. One may be sarcastic and still remain serious. When the patient cannot easily decide whether certain remarks are meant seriously or not, they become so much the more effective. Sometimes it is advantageous if the therapist can succeed in assuming a stupid role. When the patient is faced with the necessity of explaining in detail his reason for his attitude, often he readily comes to realize the senselessness of his own behavior or the purpose which he had wished to attain by means of his behavior, and which previously he had not admitted to himself.

A rather unique technique which not only yields results with amazing promptness but also affords theoretical insight into the mechanism of the "nervous" symptoms was described repeatedly by Alfred Adler and has been called by Erwin Wexburg "anti-suggestion." It consists in this: the patient is advised, under some pretext and with no implication that he is being insulted, to practice just that very thing which up to now he has apparently been fighting against; i.e., to aggravate his symptom. If one persuades a patient who cannot sleep, that it might be good for him to try to stay awake once in a while, if one tells a patient who cries all day long to cry to his heart's content—then suddenly sleeplessness and weeping cease to function so well. One can observe again and again in functional disturbances, in states of fear, compulsion-impulses, etc., that the symptom diminishes in intensity when the patient consciously tries to intensify it. It might even dis-

appear entirely if these attempts were continued for a longer period of time. However, it never comes to this, because the excessive use of any measures which are employed longer than merely for the overcoming of a temporary standstill in the therapy guarantees the failure of the therapy, and usually is answered by the patient's discontinuing the treatment.

Through anti-suggestion it can be most impressively demonstrated to the patient that his apparent battle against the symptom is not *a priori* merely useless, but actually calls forth the symptom. It is the tension which he creates through this apparent battle, be it in the organic, mental or sensory sphere, which makes possible the symptom. When he ceases to struggle and the tension abates, the symptom, too, disappears. This happens, when he attempts to intensify the symptom instead of fighting against it. When he stops fighting he attacks the foundation upon which he has constructed his symptom. In this revelation alone lies the value of anti-suggestion. It cannot help him in actually getting rid of his symptom, for the premise for this would be that he would not need the symptom any more, that his inner development had already progressed to such a degree that he could avoid any effort on the useless side of life—for such is the nature of every symptom. No trick, but merely his own growing insight and regained courage can help him to change his attitude and behavior.

How does the patient get into a situation in which logical discussion will, for the moment, no longer be effective and thus compel the therapist to resort to artifices? Freud is of the opinion that this "resistance" is always directed against the uncovering of "unconscious" sexual thoughts and experiences. He believes that this shows itself most in the "transference." The latter, according to Freud, is the patient's love for the analyst, who is for him the symbol ("Imago") of all the persons he has loved before, especially those he loved in his earliest childhood. Freud makes the distinction between a "positive transference" of affection and a hostile "negative transference," the latter originating in the revolt against this love, in hatred for the analyst. The "resistance" which jeopardizes the success of the treatment is, according to Freud, the result either of the negative or of the positive transference when the latter represents repressed erotic instincts. The "component factor capable of being made conscious and not objectionable" is to Freud the nucleus of success in psychoanalytic treatment. Is this assumption which regards a hypothetical instinctive life as the core of every treatment really necessary? Do we not everywhere see in daily life that people resist when one demands something of them which they do not like? Do we need the hypothesis of an erotic attraction or rejection between physician and patient? At the same moment when the patient resists us we know for a certainty that we are expecting something from him which he does not like. When he rejects an insight, he does so, not because of some embarrassing sexual revelation or because he hates us, but rather because he expects from this revelation a burden on his life which he does not feel strong enough to bear. He is not yet courageous enough to dare to go out into life without the protection of his devices for security, which his symptoms provided, and he is afraid of being deprived by us of these weapons. Is it not understandable that he should resist?

It cannot be doubted, however, that his attitude toward the therapist may intensify or mitigate this opposition. For between therapist and patient there exists, after all, a human community, the purpose of which is to cure the patient. If the therapist is so awkward as to insult the patient, then, as in every human community, the insulted one will revolt, possibly strike back. And the only weapon with which a patient believes he can strike the therapist is a relapse, a devaluation of the therapist's work by proving it a failure.

It is not always easy to separate a flight from health from revolt against the therapist. The patient will have some criticism of the therapist's personality to express when he revolts against the result of the treatment to date. The moment when the patient suddenly notices that his physician is, after all, not the right one is very typical. This can be observed with special clarity in the case of patients who have knowledge of other psychotherapeutic theories. For quite a while they have no doubt as to whether the method applied is right or not. Suddenly they will put the question as to whether in this or that instance another psychological school of thought might not perhaps be more correct. We can be sure that this is the moment when the patient wants to escape from the pertinence of our arguments. His search for excuses with which to destroy the foundation of the discussions proves this. I have found this supposition to be correct in every case. We find similar motivations existing when the patient suddenly brings forward political or religious objections to the therapist.

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METHODS FOR THE STUDY OF HUMAN ARTERIAL HYPERTENSION

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THE social and medical importance of arterial hypertension is demonstrated by the following figures. According to recent statistics of the Metropolitan Life Insurance Company,[1] the diseases of the cardiovascular and renal systems constitute about 50% of all causes of death in individuals over 50 years of age in the United States. Of these, Fahr[2] has demonstrated that about half are due to arterial hypertension. Arterial hypertension, therefore, is directly or indirectly the cause of death in approximately one quarter of the people over 50 years of age. One has only to consider the fact that approximately 25% of the entire population of the United States is over 50 to realize that arterial hypertension is the gravest medical problem of adult age, not excluding cancer.

None of the new surgical and medical methods of treatment have proved, as yet, to be the answer to the problem. Sulphocyanate, renal extracts, fish oil derivatives and sympathectomy are still in the experimental stage.

From the historical point of view, it is interesting that the following fundamental discoveries followed each other at intervals of almost a century: the discovery of circulation, the first method for the determination of arterial pressure in animals, the relationship between renal lesions and heart hypertrophy and, finally, the production of permanent renal hypertension in animals. Undoubtedly these long intervals of time were due to the lack of adequate methods for the study of arterial hypertension. Many methods are available today; in most instances, however, the lack of uniformity and standardization makes it impossible to compare clinical data and therapeutic results obtained by different investigators.

It is the purpose of this article to suggest a standardized program for the study of arterial hypertension in man, based on the recent experience of Peet et al[3] and on other methods which have not yet become part of the routine physical examination.

Arterial hypertension is a polymorphic disease which lends itself poorly to a systematic description. This explains the many classifications suggested, most of which, although containing many elements of truth, are too schematic, and are not comprehensive enough. A relatively simple classification of hypertension has been suggested recently by Weiss.[4] (See Figure 1.) In accepting this or any other classification one must remember that the factors described are generally interdependent and often associated in the same individual. In Weiss' figure, the hereditary and constitutional factors constitute the base of the pyramid and the endocrine, nervous and psychic factors form the sides, described in greater detail in the three adjoining triangles. It is not the purpose of this article to discuss this or any other classification; it will be sufficient to remind the reader that, no matter what its cause, be it nervous or psychic, endocrine

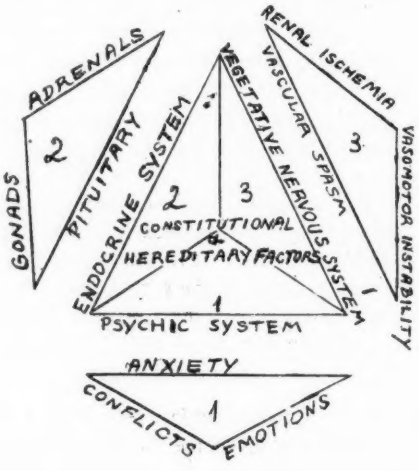

Fig. 1

or renal, hypertension is a disease of the vascular system, and the examination of this system cannot be limited, as too often is done, to the measurement of the blood pressure or of the cardiac enlargement.

The regulation of arterial pressure is the result of a delicate equilibrium of nervous and humoral forces. Among its determining factors, the pulse rate, the cardiac output, the blood volume, the blood viscosity and the elasticity of the blood vessels, are not altered in essential hypertension, unless cardiac decompensation, arteriosclerosis or other complicating factors are present. The discussion will be limited, therefore, to the last and most important factor in the regulation of arterial pressure: vascular resistance. This is composed of three principle elements[1]: the resistance due to the friction of the blood against the walls of the central arteries and their main branches,[2] the resistance opposed by the arterioles,

and[3] the resistance opposed by the capillaries. Approximate computations reveal that, even in case of extreme arteriosclerosis, the friction against the walls of the main arteries does not produce a change in arterial pressure greater than 2.5%. The resistance of the capillaries can also be disregarded, unless there is cardiac decompensation. The arteriolar resistance however, is very important, as demonstrated by the fact that, while there is practically no difference in pressure between the left ventricle and the main arteries, the pressure in the arterial loop of the capillaries is, on the average, only 30 mm of mercury. Uncomplicated arterial hypertension is due largely to a rise in arteriolar resistance. Normally the contraction of the arterioles in one vascular district coincides with a dilatation in other parts of the body and the blood can be distributed as needed without an increase in total vascular resistance or in arterial pressure. If, however, the vascular system is subjected to abnormal vasoconstrictor stimuli, a large proportion of the arterioles of the body may constrict at the same time, producing a rise in blood pressure. If the abnormal stimuli are repeated with sufficient intensity the periods of high blood pressure mechanisms begin to fail, a vicious circle may be established and hypertension may become permanent (Figure 2). If, as often occurs, the functional spasm of the arterioles is followed by hypertrophy of the arteriolar wall, the disease may become irreversible. The existence of arteriolar spasm in hypertensive patients has been demonstrated conclusively by Pickering,[5] by Prinzmental and Wilson[6] and others in the skin, muscles, brain, kidney and in the territory innervated by the splanchnic nerves. It has also been shown that hypertension of long duration is almost always associated with permanent hypertrophy of the arteriolar wall.

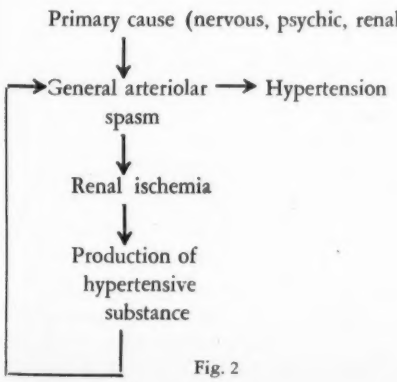

Fig. 2

A study of the circulation, as outlined below, has proven of value in the prognosis of hypertension and in the evaluation of the therapeutic results.

METHOD

It is recommended that before any therapeutic attempt the patient be classified according to the following criteria:

1. History and physical examination, with special attention to nervous, mental, endocrine diseases and eclampsia.

2. Examination of the subjective symptoms and physical incapacitation. To obtain this information the physician must rely on the statements of the patient and great care must be used in the evaluation of the data. The subjective symptoms are classified as follows: Absent, Mild, Moderate, Severe. The incapacitation as: Total, Severe, Moderate, Mild, Absent. After therapy the subjective symptoms are classified as: Absent, Improved, Unchanged, Worse. The unreliability of such classification is obvious.

3. Examination of the heart. The cardiac function is classified according to the criteria of the American Heart Association,[7] as follows: First group, patients with asymptomatic heart disease without limitation of physical activity; Second group, patients with mild cardiac symptoms, such as fatigue, palpitation dyspnea and pain appearing after normal physical activity; Third group, patients with more severe cardiac symptoms which can still be controlled by rest; Fourth group, patients with signs of cardiac decompensation forced to complete inactivity. The degree of cardiac enlargement is measured by means of the teleoroentgenogram and the orthodiagram. The heart is considered enlarged if the area is at least 15% larger than normal, weight and height of the individual being taken into account. The electrocardiogram is judged according to the degree of deviation and abnormalities of the T wave. The

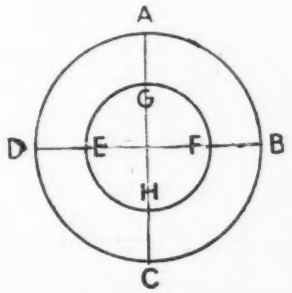

Fig. 3

cardiac function is considered improved after therapy only if the change is sufficient to determine a change in group; the cardiac hypertrophy, only if there is a change of at least 10% in the area. The electrocardiogram is classified as "Improved" or "Worse" only if the change is beyond doubt; otherwise it is classified as "Unchanged."

4. Examination of renal function. The patient is submitted to intravenous and, if necessary, retrograde pyelography to rule out the presence of renal stones, hydronephrosis, pyelonephritis, congenital malformations or other surgical lesions. History and analysis of the urine permit, in most cases, the ruling out of acute or chronic glomerulonephritis. The following other determinations are made: a) NPN, which is considered normal if below 36%; b) Urea clearance, which is considered normal if it is 70% or more of the average normal value; c) Urine concentration test, which is considered normal if the specific gravity of the urine is at least 1.029 after 18 hours without food or water. A change of 0.003 in specific gravity of urine is considered significant. The urea clearance is classified "Returned to normal" if it rises to or above the normal value, "Improved" or "Worse" if there is a change of at least 15%, otherwise "Unchanged."

The above described routine examination, whenever possible should be supplemented by the study of the renal circulation, as it is known that hypertension may, and very frequently does, exist without damage to the excretory function of the kidney. The study of renal circulation in man has been made possible by Smith and his collaborators[8] by means of diodrast and inulin clearance. The details of the technic are described in Smith's papers. It will be sufficient to say that the method is based on the fact that diodrast is completely cleared from the plasma by the kidney if its concentration is below 5 mg per 100 cc. Its clearance is therefore a measure of the plasma circulating through the active renal tissue. The renal plasma flow naturally varies according to the size of the organ. In order to compare results obtained in different individuals the plasma flow is expressed in relation to the so called tubular mass. The tubular mass is measured by the maximal amount of diodrast which the renal tubules are capable of excreting when working at capacity, the concentration of diodrast in the plasma being 20 mg per 100 cc. or more. The ratio between plasma flow and tubular excretory mass varies within relatively narrow limits in normal individuals. Inulin is eliminated only by filtration; its clearance is therefore a measure of the filtration rate and gives useful information about the glomerular vessels. The determination of diodrast and inulin clearance has been recently simplified and can be performed in any hospital laboratory without the need for special equipment[9].

The determination of diodrast and inulin clearances in 22 hypertensive patients and in 20 normal subjects gave the results reported in Table 1. Renal plasma flow in hypertensive patients is about half of normal. The filtration rate is less than normal;

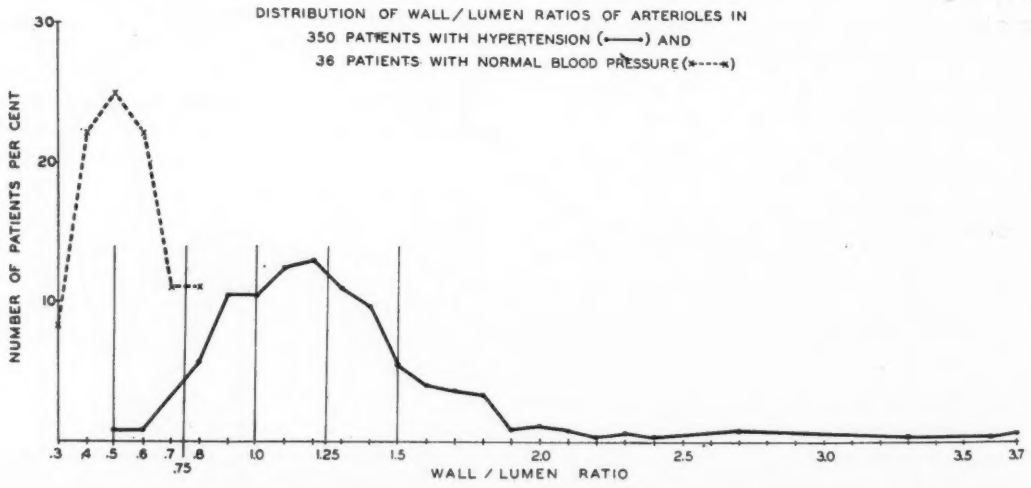

Fig. 4

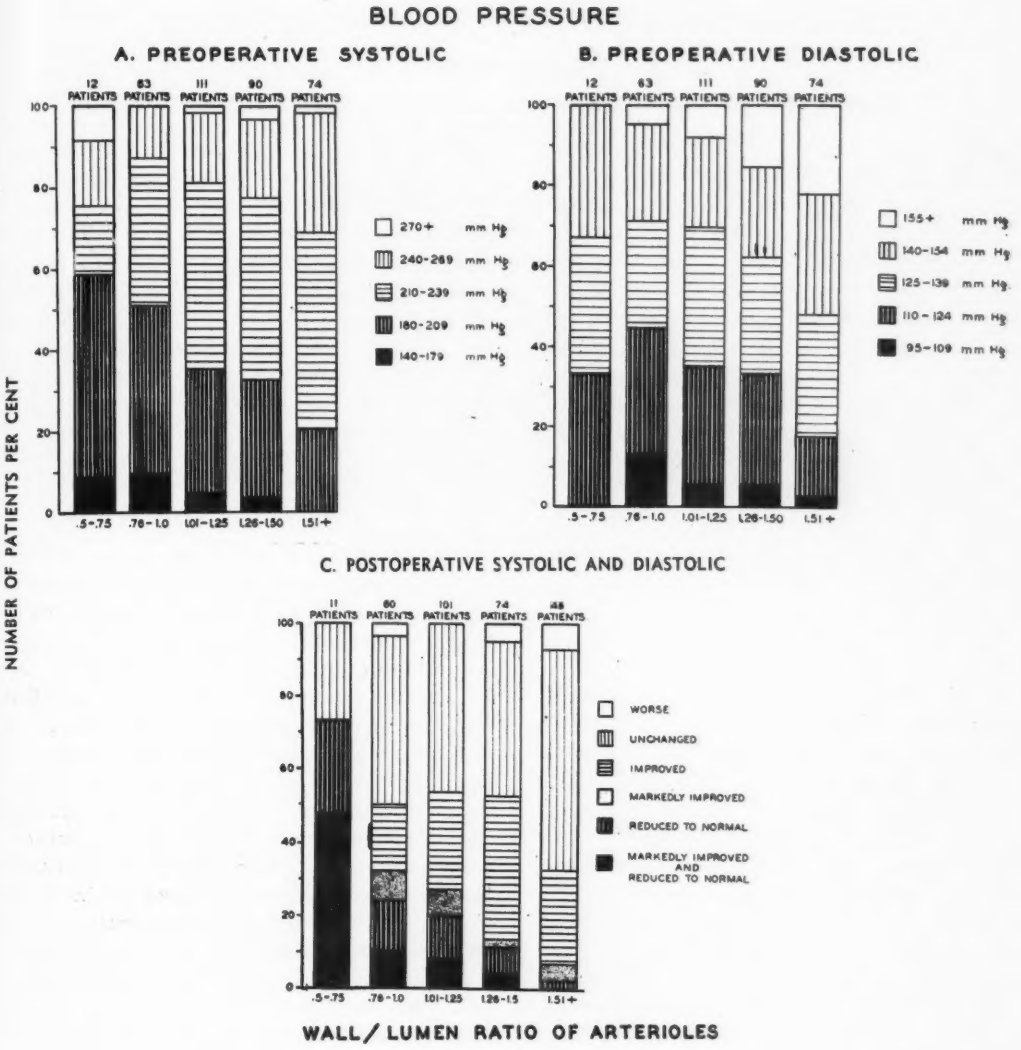

Fig. 5

the reduction in filtration rate, however, is not in proportion with the reduction in plasma flow, so that the fraction of the plasma cleared by filtration (filtration rate/plasma flow) is higher than normal. The tubular excretory mass is low. The average ratio of the plasma flow to the tubular mass, which represents the plasma flow per unit of tubular mass is reduced, whereas the ratio of inulin clearance (filtration rate) to tubular mass is increased. Similar results were obtained by other investigators.[10, 11]

The results show that arterial hypertension is associated with renal ischemia and high filtration fraction. The filtration rate is kept adequate despite the reduction in blood flow. If the reduction in blood flow is very severe, this compensatory mechanism is insufficient to prevent signs of renal failure, such as increased NPN and decreased urea clearance.

To determine the prognostic value of diodrast and inulin clearance, the patients were arbitrarily arranged according to their renal plasma flow and divided into two groups. The first group was comprised of patients whose renal blood flow was less than 535 cc/min. (the average value of all patients); the second group contained the patients whose renal blood flow was higher than the average value. The

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THE TREATMENT OF SURGICAL COMPLICATIONS

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THE surgeon has come a long way from the early days of heroic medicine, and after having passed through the succeeding Anatomic and Pathologic eras has reached his greatest triumphs in the present Physiologic period. Yet in spite of the tremendous advances that surgeons have made during each of these successive periods, morbidities and deaths still occur even after the best of adequate pre-operative, operative, and post-operative care. Most responsible for these are such complications as Shock and Hemorrhage; Wound infection and disruption; Ileus, Intestinal obstruction and Peritonitis; Pulmonary embolism, Massive collapse of Lung and Pneumonia; Thrombophlebitis and Phlebothrombosis; and Hepatic, renal, and cardiac failures. To avoid such disasters, early recognition and prompt treatment of these complications constitutes the important part of adequate post-operative care. This does not only fall to the lot of the surgeon but to the alert surgical resident and interne as well.

Shock and Hemorrhage:

The typical picture characterized by prostration, pallor, rapid thready pulse, apathy, falling blood pressure, and subnormal temperature is easily recognized and is best combatted by transfusion of the appropriate fluids—plasma, serum, or blood. If these are not immediately at hand, saline, glucose, and some of the gels such as acacia, petin, gelatine, and isinglass may be employed. The objection to the saline and the glucose is that they do not stay in the blood stream and therefore do not maintain the circulating volume; the objection to the gels is that they produce necrosis and clumping in the spleen and liver when used in adequate amounts and in isotonic concentration, as was shown by the recent work of Meyer and Volk and others. As a result of this clumping several deaths have occurred. Plasma therefore is to be preferred, as it needs no typing, but enough must be given if success is to be attained. A general rule that holds well is to give 10 cc. of plasma per kilogram body weight and to double this amount if necessary. If blood loss is the cause of the shock, the plasma transfusion should then be followed by a transfusion of 500 cc. of whole blood and the result checked with subsequent blood counts and hemoglobin determination. Anoxia often accompanies shock and is due to the diminished ability of the blood to carry oxygen to the tissues. Its presence is noted by cyanosis and is most effectively treated by oxygen inhalation using the Boothby mask. Should concealed bleeding from a large vessel be suspected such as occurs from a bleeding cystic, uterine, superior thyroid, or large vessel of the extremity, packing is inadvisable because it may not control the bleeding and fatal blood loss can result, or further damage can be done from the tamponading effect on the collateral circulation, as in an extremity, with threatened gangrene to the distal part. Under such conditions one must not delay in attempting to find and ligate the bleeding vessels, but only after the primary treatment for shock is instituted.

Wound Infection and Disruption:

The intentional wound made by the surgeon in a clean operation should remain clean and should heal *per prima* with a minimum of scar and a maximum of strength. This means a clean field prepared by cleansing with soap and water and shaving off all the hairs. Careful scrubbing (usually 10 minutes) of the hands and forearms of the surgical team, who wear sterile gloves, gowns, and masks to cover both nose and mouth, and who are disciplined to keep their mouths shut during the operation, prevents outside infective material from getting into the wound. Sterile towels clipped to the edges of the wound likewise keep the sweat of the surrounding skin of the patient from seeping into the wound. Careful hemostasis and avoidance of ligatures *en masse* that may strangulate large masses of tissue avoids creating a suitable culture media, so to speak, that may prompt the growth and activity of pathogenic organisms. These are always normally present in the deeper layers of the skin.

Wound disruption fortunately is a rare occurrence and most always involves the abdomen. It is a most annoying post-operative experience to have the nurse or interne call you because they have noted some large mass that appears like omentum or intestines to be lying beneath the dressings of the abdominal wound. Fortunately these eviscerated organs can be replaced into the peritoneal cavity without much fear of peritonitis if this is done promptly and aseptically followed by a careful through and through suture of the wound. We know now that disruption can occur in the clean wound which has been carefully sutured

layer by layer with accurate approximation, that it can occur in the patient free from a luetic infection, and that it can occur in the patient who has not coughed, sneezed, or strained by vomiting post-operatively. Recent studies have revealed that a low value in the vitamin C blood level below 1 mg. per cent fails to produce an effective fibrogenesis with subsequent poor healing at the wound site. This leads to wound disruption. The daily post-operative administration of vitamin C in 100 to 300 mg. amounts given by the oral or intravenous route promptly stimulates fibrogenesis and results in a good scar with strong union.

Ileus, Peritonitis, and Intestinal Obstruction:

Every surgical procedure on the intestinal tract is followed by some degree of ileus or intestinal paresis due to a local shock of the neuro-muscular portion of the gut. Fortunately this mild ileus clears up after the second or third post-operative day. With excessive and rough handling of the intestines, forceful retraction, excessive packing away of the intestines to clear the operative field, undue cooling of the peritoneal contents, prolonged anesthesia, a permanent and progressive ileus of the entire alimentary tube may occur. As a result a tremendous abdominal distention with upward displacement of the diaphragm occurs, and should this not be relieved, a fatality by cardio-respiratory failure results due to the compression. Occasionally ileus is seen in the simple surgical case where operation is done on some portion of the abdominal wall with an entry into the peritoneal cavity as for instance in the repair of an inguinal hernia. Prompt treatment of ileus consists of gastrointestinal suction using the Levin tube which quickly relieves the distress due to the gaseous distention. Should this distention be in the colon, then insertion of a rectal or colon tube and injection of a return flow enema will give the desired relief. To stimulate peristalsis 200 cc. of a 3% NaCl solution is given intravenously, and if this fails, a spinal anesthetic or a paravertebral sympathetic block of the lumbar region may be given. This abolishes the inhibitory sympathetic nerve control and prompt intestinal evacuation usually follows. My experience with prostigmin both pre-and post-operatively for preventing ileus, giving it intramuscularly every 4 hours for at least 3-5 days, has failed to convince me that it is of great value. To employ it routinely as some recent publications advise is unnecessary. At the Illinois State Prison where I had the opportunity to try prostigmin on a small group of surgical patients it was revealed that those not receiving prostigmin got along better than those who had been given it. Very often the patients complained of abdominal pain after its injection, and it is possible that this was due to local spasms of the intestinal tract produced by the drug. In this respect it could even be harmful.

Intestinal Obstruction:

Occasionally an acute mechanical intestinal obstruction may follow intra-abdominal operations due to the formation of adhesions by organization of fibro-plastic exudate which kinks some part of the gastrointestinal tube. I recall one very vivid case in a young girl of 20 on whom I performed a simple cholecystectomy. For the first 48 hours she did well and then developed the most violent colicky pain in the upper abdomen associated with projectile vomiting of gastric secretion. Her facies were drawn, she was apathetic and preferred to be left alone and refused all liquids and nourishment allowed her. No distention was present and it was decided that she had a gastric obstruction. After considerable persuasion, a barium meal was given and observation under the fluoroscope revealed a high gastric obstruction. In spite of much wretching, the barium (because of its weight) stayed in place. Needless to say the patient looked sick, and then with dramatic suddenness the patient was relieved of all distress and stated that she suddenly felt as if "something tore within," and the pain, fullness, and nausea disappeared. No doubt the weight of the barium meal and gentle pressure applied to the abdominal wall during the fluoroscopy gradually tore these early, unorganized fibro-plastic adhesions. A plate of the abdomen about 2 hours later revealed good emptying of the stomach. Her further post-operative course was uneventful. This method of relieving early post-operative gastric obstruction was suggested to me by Dr. A. Straus of Chicago and is applicable only when extra-gastric surgery is done. On the other hand, as a word of warning, this method of treatment would be dangerous and improper where the obstruction was suspected to be due to post-operative adhesions of the intestine proper. In the latter, vomiting, anorexia, and colicky pains also occur plus a gradual distention and early passage of a varying number of liquid stools. Prompt intubation with Wangensteen suction is often all that is necessary to deflate and to place the bowel at rest and allow the accompanying edema produced by the inammation and hyperperistalsis to subside. Should obstruction persist, surgical intervention for relieving the occluded bowel can then be performed with greater safety and with ease. As for

prophylaxis, careful preparation using no cathartics and not too vigorous a cleansing with enemas, preliminary gastric lavage and Levin suction for distention are most important. The surgical technique calls for minimal handling lest the serosal surfaces be damaged and the intestine shocked. Careful hemostasis and peritonealization of all raw surfaces, taking time to repair all large rents in the mesentery and omentum and care to avoid *en masse* ligatures with heavy suture material are the other important points to be remembered. The latter has been known to produce an abundant outflow of inflammatory exudate that may organize into obstructive bands and membranes, or cause loops of bowel to adhere with resulting obstruction.

Peritonitis:

Peritonitis is still the most dreaded of all abdominal complications and fortunately in the clean cases is rarely seen. Advances made in the past for its prevention have largely been through careful preparation, early operation on the lesions that may produce peritonitis such as perforations and appendicitis, decompression by intubation, and careful surgical technique that avoids intestinal leakage. The intra-peritoneal vaccination of Bargen has been discarded, for it causes too intense a peritoneal inflammatory reaction rendering suture of this structure difficult. The use of polyvalent anaerobic and B. Coli sera advocated by Weinberg and Steinberg, although favorable, is not necessary. In badly suppurative appendicitis when it is feared that a suppurative pylephlebitis might occur, I have placed 40 cc. of cololysate liquid routinely into the peritoneal cavity with good results. The most recent and the most important contribution has been the intraperitoneal use of the sulfonamides. Of these sulfanilamide and sulfadiazine are the best for B. Coli, Streptococci, Staphylococci, and Gonococci infections. When placed in the peritoneal cavity they should be sprinkled in a finely powdered form and not be allowed to gather in a clump. Not more than 5 to 10 gms. should be introduced lest toxic effects result in the form of cyanosis, hematuria, and anuria. Additional sulfa drug is given intravenously in doses to keep the blood level up to 8-9 Mgm. percent. Always with sulfa administration two things must not be forgotten; these are: giving plenty of fluids (3000 to 4000 cc. per day) and alkalinization of the urine to prevent hematuria. Without alkalanization, acid hematin crystals are formed from the hemoglobin of destroyed red blood cells, and these crystals then plug up the glomeruli, resulting first in a hematuria and, if very severe, in complete anuria that may lead to uremia and death. Recently a new sulfa preparation has been introduced that is not absorbed from the intestinal tract and that destroys the B. Coli organism. This drug is sulfaguanidine. It renders the colon free of pathogenic bacteria and has remarkably reduced the mortality in large bowel surgery, enabling the surgeon to do more and more primary resections and anastomoses of the colon. Dixon advocates that the drug be given orally for three successive days preoperatively, and employs 12 Gms., 8 Gms., and 6 Gms. respectively. No drug is given postoperatively. In a series of some 400 cases he has had a mortality of less than 2%. In children, pneumococcic peritonitis is often encountered, with its characteristic pale thin green exudate. By typing this exudate, the specific type of pneumococcic serum can then be given along with the sulfadiazine.

Pulmonary Complications:

Respiratory difficulties are always dreaded because they produce one of the most frequent causes of post-operative death and because the accompanying cough adds greatly to the development of post-operative hernia or wound disruption. Several different conditions may arise and the most common of these are: massive collapse of the lung, pneumonia, and pulmonary embolism. It was formerly believed that inhalation anesthesia was responsible for these complications, but their occurrence under local and spinal anesthesia disprove this. After every major abdominal operation there is a shock to the thoracic and abdominal muscles and to the diaphragm with diminished ventilation and lowered vital capacity of the lung. This favors development of pulmonary complications.

Massive Collapse of Lung (Atalectasis):

This occurs as a rule in the first 72 hours and is characterized by a high fever (102-105 degrees), thick tenacious sputum, diminished breathing, and dullness of the involved lobes and hyper-resonance of the opposite side, with shifting of the cardiac dullness toward the affected side. The best explanation of this condition is that a mechanical obstruction of the bronchus occurs by accumulated secretions in the tracheo-bronchial tree. If a small bronchiole is occluded, only a lobule is affected, if a larger bronchus is occluded, a lobe is involved, and if it is the main bronchus, a massive collapse of the entire lung on that side results. All of the air is then absorbed from the lung distal to the occlusion, resulting in its collapse or atalectasis. Preventive treatment is directed to produce hyper-ventilation. This consists of giving

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THE PRESENT STATE OF THE SALMONELLA PROBLEM

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EVER since Salmon and Smith in 1885 first isolated a member of that group of organisms later to be known as "Salmonella," confusion has resulted from the diversity of their source, and lack of unity in nomenclature. These organisms appear in a variety of diseases in both man and animals, but were recognized as belonging to one great group as early as 1897 by Smith and Stewart.

To the casual observer it may appear that Salmonella strains present an appalling collection of meaningless names. To the initiated, however, the explanation is comparatively simple. Original workers adopted the procedure of naming Salmonella according to the animal source from whence the organisms were isolated, i.e. *S. anatum.* Later investigators attempted to use a system of nomenclature which would describe the disease produced, i.e. *S. abortus-equi.* As more and more Salmonella were discovered, so much confusion arose from the varying nomenclature that the Salmonella Subcommittee of the International Association of Microbiologists decided to adopt a definite schedule wherein a new Salmonella type would bear the name of the locality, country, or colony where it was found. Examples of Salmonella named according to this rule are *S. illinois, S. kentucky, S. tel-aviv, S. bareilly* and *S. panama.* If the name of the country or locality has already been given to a Salmonella, the name of the hospital where the organism was isolated is used, i.e. *S. bispebjerg* and *S. virchow.*

It is impossible in this limited space to give the full list of some 120 Salmonella organisms. The reader who is interested in studying such a list is referred to Gradwohl[1], Bornstein[2], Edwards[3]. The following, however, are the Salmonella species which are of greatest importance, presented according to the group in which they occur.

Group "A": Salmonella paratyphi, formerly called B. paratyphosus A, causes a typhoid-like fever and occurs mainly in the tropics. It is rarely found in the United States. Many persons infected with it become chronic carriers. The mortality is about 1%.

Group "B": S. schottmuelleri, formerly called B. paratyphosus B, is a widespread cause of typhoid-like fever, but occurs also in gastroenteritis and in suppurative processes. Found in numerous chronic carriers, it is a very common organism. The mortality rate is about 2%.

S. typhi-murium, called S. aertrycke in the older nomenclature, is the commonest incitant of Salmonella gastroenteritis, septicemia and localized infections in the United States. The organism is harbored by rodents, fowl and swine as well as human carriers. The mortality is about 6%. A variant of this species of Salmonella, known as S. copenhagen, has a mortality rate as high as 25%.

Group "C": S. hirschfeldii, formerly known as B. paratyphosus "C," is the source of a typhoid-like fever in the tropics. It is not present in the United States.

S. cholerae-suis, formerly named S. suipestifer,, has two varieties. The first, called "European," is encountered mostly in America. The second, called "American," is rarely found outside of Europe. This Salmonella bears a strong similarity to S. hirschfeldii, but differs in its fermentation of certain carbohydrates and its lack of one portion of that organism's antigenic structure. S. cholerae-suis causes septic fever and localized abscesses in human beings. It is considered one of the most dangerous Salmonella, having a mortality rate as high as 25%

S. montevideo causes mainly gastroenteritis in humans, but may also instigate a localized disease. It occurs in several animal species. The mortality rate is low.

S. oranienburg differs from the above only slightly in its antigenic structure. It is found mainly in fowl, but is capable of inciting epidemics in man with a mortality rate as high as 10%.

S. newport is found in all clinical forms of Salmonella infection. This Salmonella is present in many animals, but is also frequently spread by human carriers. The mortality rate is 6%.

Group "D": Eberthella typhosa, the typhoid bacillus, is now classified with the Salmonella, in England actually being called "Salmonella typhosa." It differs from the other Samonella strains in forming only acid, not gas, from carbohydrates. The mortality rate is about 8%.

S. enteritidis, formerly named B. gaertneri, produces gastro-enteritis, and also septicemia and localized lesions. It occurs in many animals and human carriers. The average mortality is 4%.

S. sendai usually causes a typhoid-like infection of low mortality. It is found mainly in the tropics.

S. pullorum and its non-gasforming variant, S. gallinarum, are more interesting from the taxonomic than from the practical point of view.

Group "E": S. give is an organism found in fowl and in human carriers. It incites gastro-enteritis.

S. anatum causes gastro-enteritis, rarely septicemia or a local abscess. It is harbored by several animals and by human carriers. The disease is rarely fatal.

S. senftenberg may be present in various animals and in human carriers, instigating a gastro-enteritis. No fatal cases have been described in the literature.

Group "F" or "Further Organisms": These occur rarely. The most interesting species of this group is S. ballerup.

Research workers pay much attention to the following species because of unusual characteristics of their antigenic behavior: S. salinatis, S. sandiego and S. abortus-equi.

Salmonella includes a large and variable group of micro-organisms. They have been most accurately defined by a subcommittee of the International Association of Microbiologists as "A large genus of serologically related, gram negative and non-sporing bacilli; 0.4-0.6 mi. × 1-3 mi. in usual dimensions, but occasionally forming short filaments; showing, with certain exceptions, a motile peritrichous phase in which they normally occur; in fact adhering to the pattern of E. typhosa in staining properties and morphology. Failing to ferment sucrose or to clot milk, and rarely fermenting lactose, liquefying gelatin or producing indol, they regularly attack glucose with, but occasionally without, gas production. All the known species are pathogenic for man, animals, or both." This definition itself, with its numerous exceptions, demonstrates the difficulties encountered in the classification of this group according to their biochemical activities.

A sound basis for the classification of Salmonella was finally furnished by Kauffman and White who identified the varying species by means of their antigenic structure.

Today the investigation of Salmonella strains proceeds according to a well established schedule of antigenic analysis. Most Salmonella contain several "O" and "H" antigens and some also carry the antigen designated as "Vi."

The "O" antigens are somatic, heat and alcohol stable, and are named by means of Roman numerals, as I, II, III, IV, etc.

The "H" or flagellar antigens are thermolabile, but formalin stable, and can be found only in motile members of the genus. Organisms containing these antigens usually appear in two phases, one of which is called the "first" phrase and is labeled with small letters from "a" to "z" and finally "z_1", "z_2", "z_3", etc. The other phase of the flagellar antigen, the "second phase," is designated according to Arabic numerals as "1", "2", "3", etc.

Many Salmonella types lack the first or second flagellar phase.

When the "Vi" antigen was discovered by Felix and Pitt[4] it was supposed to be associated with virulence. Its presence in the bacterial body increases the inhibition of agglutination with "O" antisera. This antigen is complex in nature and can be analyzed with the aid of bacteriophages.

It has been found that many organisms not belonging to the genus Salmonella may reveal antigens common to this group. Kauffmann[5] was willing to accept any organism into this genus which showed Salmonella antigens. It has been found that many coliform organisms have such antigens, mainly I, V, VI, VII, XII, 1, 5, and even Vi. Paracolon bacilli often possess the antigens I, II, V, VI, XII, z_4, z_{23}, and z_{24}. Occasionally even organisms which have nothing to do with the family and tribe to which Salmonella belong reveal such antigens. This is true of *Pasteurella psuedotuberculosis* which harbors part of antigen IV, *Pasteurella avicida* containing the Vi antigen, and the 35th type of pneumococci which reveals antigen XVII. It is generally known that antigens XI and XIII are shared by *Shigella paradysenteriae,* thus presenting a considerable obstacle in the classification of these organisms.

Serologically and epidemiologically *E. typhosa* belongs in group "D" of Salmonella, but because it does not form gas from dextrose and mannitol, as do most Salmonella, it has been put into a separate genus, *Eberthella.* Other organisms antigenically belonging to the Salmonella present similar difficulties. Because "Shigella" gallinarum is non-motile and does not form gas, it was classified with the Shigella. *Salmonella pullorum* is even more problematical because it forms gas, but is non-motile. *Salmonella dar-es-salaam* deviates from the accepted standards in its liquifaction of gelatin. *S. typhi-suis* fails to ferment mannitol.

In order to clear the taxonomic situation and facilitate the proper classification of these organisms demonstrating aberrant biochemical properties, Amer-

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ESSENTIALS OF DIABETIC MANAGEMENT

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THE REGIMES to be presented in this brief are based upon the contributions of Drs. Samuel Soskin and Rachmael Levine, of Michael Reese Hospital, Chicago, to the field of carbohydrate metabolism. We have used their interpretations of the patho-physiology of diabetes mellitus as guiding principles in clinical management.

DIABETIC COMA: (treat dehydration, shock, and acidosis). Essential points in the *diagnosis:*

a) Coma; Kussmal breathing; "fruity" odor of breath.

b) Marked glycosuria and acetonuria (the diagnosis cannot be made in the absence of acetone in the urine).

c) Hyperglycemia (greater than 160 mg. %—renal threshold).

d) Low CO_2 combining power, usually 30 or below (normal values 55 to 75).

Treatment:

1. Insulin: 200 units initially; 100 units (child 50 units) insulin hydrochloride in first glucose flask; then 100 units insulin-HCl subcutaneously (child 50 units); give 70 units (child 40 units) insulin-HCl q. 2 hours.

2. Glucose: (500-900 Gm. in 24 hours); administer I-V as 10% glucose in *normal saline.*

3. Hartman's Solution: (sodium lactate); to aid in combatting the acidosis (if CO_2 combining power is very low), 1000 c.c. after 2000 c.c. of glucose has been given.

4. Watch cardio-vascular system: give cardiac stimulant (coramine, metrazol, caffeine) p.r.n., but never give epinephrine; it is here that constant attention is so necessary.

5. Test urine every 4 hours: when urine is acetone-free (patient comes out of coma), gradually reduce, but *never* discontinue insulin; continue I-V fluids.

6. When patient is well out of coma and is able to take fluid by mouth: orange juice 10 oz. with 1 tablespoon sugar q. 2 hours (alternate with milk if patient desires); give 40 units (child 20 units) insulin-HCl q. 4 hours (if reactions occur reduce the amount); continue this prescription until 6 successive 4-hour urine specimens are acetone-free.

7. After the urine is acetone-free, immediately calculate the diet and insulin dosages along the principles outlined under Diabetic Maintenance. It is to be emphasized that the patient should be put on the maintenance diet *immediately,* and that, in our experience, "low starting diets" are never necessary.

If the above regime is strictly followed, and if the patient has not been in coma for more than a few hours, a return to consciousness may be expected within 6 or 8 hours.

It is always necessary, especially in older patients, to distinguish between hepatic hyperglycemia* (due to liver damage), and true diabetes. The essential diagnostic point is that in the former, acetone is seldom found in the urine (and when found is usually due to concomitant infection).

In cases of deep and prolonged coma (10-12 hours), it may be necessary to give 25% glucose I-V immediately (through radial veins), covered by insulin.

DIABETIC MAINTENANCE REGIME

1. Make up diet on the basis of the following principles:

a) caloric need (e.g. 30 C/kg. body weight for adult doing office work).

b) ratio of carbohydrates to fats at least 3 to 1.

c) proteins, 1.5 Gm./kg. body weight (child 2.5 Gm./kg. body weight).

d) fats, 60 to 75.

Suggested 2800-3000 calorie diet:

Carbohydrates	400-500 Gm./24 hours.
proteins	100
fats	60-75

2. Regulate insulin dosage empirically, i.e., according to patient's individual need and reaction.

Start with 20 units insulin-HCl t.i.d.; a.c.; et 15 units h.s. (with a night feeding of orange juice and sugar which is in addition to regular diet).

If sugar spills heavily in the urine, increase insulin dosage (or decrease dosage if urine is sugar free, but *never stop* insulin).

(Continued on page 27)

*Diabetes Mellitus and Hepatic Dysfunction Hyperglycemia; S. J. Taub, W. H. Shlaes, L. Rice; in press.

Alumni News

1898—Dr. M. H. Pauly of Chicago extends his very best wishes to the students of the School. Dr. Pauly is at present engaged in general practice here in Chicago.

1901—We extend our sincerest regrets to the family and friends of Dr. A. Christensen, of Chicago. Dr. Christensen died on April 5, 1944, respected and revered by all those who knew him.

Dr. F. P. Thompson is specializing in Eye, Ear, Nose, and Throat. Dr. Thompson is located on Chicago's South State Street.

1904—Word comes to the QUARTERLY that Dr. O. L. Ballenger is engaged in general practice in Chicago Heights, Illinois.

Dr. Frederick G. Roberts sends his best wishes from Chicago's West Side.

1906—We are sorry to hear that Dr. Carl R. Peterson of Chicago has retired because of poor health. We wish him the best of luck and a very speedy recovery.

Dr. William J. Schaffer is engaged in general practice in Chicago.

1908—Dr. John A. Elliot of Chicago is busy with his general practice.

1910—Many thanks to Dr. Raoul L. Vioran of Akron, Ohio, for his compliment to the QUARTERLY. Dr. Vioran is a general practitioner in Akron.

1918—Dr. Julius I. Mandel is at the present time engaged in general practice in North Chicago.

General Medicine and Surgery keeps Dr. C. G. Sachtleben of Cottage Grove quite busy.

1919—Word comes from Dr. A. J. Offord of Chicago, that he is doing well in his practice. Dr. Offord is specializing in Internal Medicine.

1920—Greetings from Dr. Joseph V. Sterba, President of the Savoy Drug and Chemical Company, of Chicago.

Dr. M. A. Sauthmick intends to retire to a four acre farm in Montana, July 1. Dr. Sauthmick has served as Curator of the Museum and Instructor in the Department of Pathology at the University of Chicago since his graduation.

Director of the Blood Bank at Cook County Hospital, past President of the Irving Park Branch of the Chicago Medical Society, and present Secretary of the organization are only a few of the many activities of Dr. H. L. Wallin. Dr. Wallin is engaged in general practice.

1921—Dr. William A. Hoffman is specializing in Eye, Ear, Nose and Throat. Dr. Hoffman's office is located here in the City.

1922—Thanks to Dr. Paul G. Papsdorf for his check to the QUARTERLY. Dr. Papsdorf is very busy in his practice of Oral and Plastic Surgery, and Ear, Nose and Throat work. He is a dairy farmer in his few spare moments.

Best wishes from Dr. Louis E. Schaeffer who is successfully engaged in Industrial Surgery and General Practice here.

Dr. Charles G. Slanec is engaged in Eye, Ear, Nose and Throat work in west Chicago.

1924—We of the Quarterly extend our very best wishes for a continued successful practice to Dr. Carolina M. Gentile. Dr. Gentile is a physician and surgeon in Chicago.

1925—Best wishes to Dr. James Aubrey Megahy of Chicago. Dr. Megahy is engaged in a general practice.

Dr. Al K. S. Margeris is busily engaged in general practice. His office is located in south Chicago.

Dr. Albert O. Stephenson is at present Associate Medical Officer at the Chicago Signal Corps Depot. Dr. Stephenson continues his general practice while engaged in this important work.

Dr. Jesse J. Anderson has retired to Jacksonville, Florida. "Anything new for high blood pressure?" he inquires.

1926—Now engaged in Neuro-psychiatry, Dr. Matthias Marquardt is practicing at the Augusta State Hospital, Augusta, Maine. Dr. Marquardt is a member of the Maine Medical Association, The American Medical Association, New England Psychiatric Association, and the American Psychiatric Association.

Our heartfelt sympathy to the family and friends of Dr. John P. Scanlan. Dr. Scanlan died of cerebral hemorrhage on March 10, 1944, after many years of devoted service to the community and his profession.

Dr. Frank Sadowski, of Chicago, is busily engaged in the general practice of medicine and surgery. Dr. Sadowski's two sons, Frank and Bob are now in the armed forces. His daughter Margaret is a student of music at Northwestern University, and manages to devote most of her spare time driving an automobile

Dear Alumni: We should like to supply each and every one of you with a copy of each issue of the Quarterly as it comes out, and we, in cooperation with the Office Staff, attempt to do so. However, a considerable number of copies of each issue are invariably returned because of incorrect addresses. We ask you to inform us promptly of any changes of address, whether civilian or military, so as to insure your receiving your copies.—The Editor.

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for the Red Cross. We wish to extend our sincerest wishes for the speedy return of Dr. Sadowski's two sons and of all those men and women now serving our country.

1928—Dr. Alfred E. Slawinski is an associate on the staff of St. Mary of Nazareth Hospital. He is engaged in general practice in Chicago.

Dr. John Walsh Williams is in general practice and Gynecology here in Chicago.

1930—Dr. S. T. Richmond informs us that he is now engaged in general practice here in the City.

1931—From Onarga, Illinois, comes greetings from Dr. Armen N. Yazarian who is now engaged in general practice. Dr. Yazarian served his Medical Internship at St. Margarets Hospital in Indiana in 1941. The following year he interned in Neuropsychiatry at State Hospital, by certificate. Since then Dr. Yazarian has been busily engaged in general practice in Dixon, Illinois; as attending Neuropsychiatrist at the State Hospital in Peoria, Illinois; as a Civilian Neuropsychiatrist for the United States Armed Forces Induction Stations in Peoria and Chicago. Today Dr. Yazarian is engaged in general practice in Onarga, Illinois, and is a member of the Iroquois County Medical Society.

1932—Dr. Evelyn A. Rinallo-Neufeld of Chicago is busy with her general practice.

Our best wishes to Dr. Frank Rojek of Chicago in his continued work.

Dr. Leonard J. Houda informs us that he is at present engaged in general practice.

1933—Dr. John J. Ringa writes us that he has recently completed a post-graduate course in Surgery. Dr. Ringa is engaged in the General Practice of Medicine and Surgery in Chicago.

1934—Dr. Charles H. Matthei of Chicago sends his greetings to the Quarterly.

Best wishes to Dr. O. R. Zinkle of Carthage, Illinois. Dr. Zinkle is engaged in general practice.

1935—Our heartiest congratulations to Dr. Phillip O. Shultz, who informs us that he is the proud father of a baby boy. Dr. Shultz has twice offered his services to the Armed Forces, and is at present engaged in private practice.

Many thanks to Dr. James M. Wall for his very nice comments on the Quarterly. Dr. Wall is practicing in Chicago.

1936—Dr. Sydney C. Rosenfeld of Irving Park writes us that he is engaged in general practice.

We are pleased to hear that Dr. M. M. Corbett is Medical Director at the Illinois division of Bendix Aviation Corps. Dr. Corbett is an Industrial Surgeon and has a brother, Bob Corbett in the Junior Class at the Chicago Medical School.

1937—Lt. P. E. Wolfe of the U. S. Army Medical Corps, informs us that he is stationed at Fort Custer, Michigan.

Dr. J. E. Segraves is located at Oak Park, Illinois, where he is specializing in bone, joint, and traumatic surgery.

1938—Dr. J. A. Sanfilippo writes us that he is engaged in general practice in Chicago.

Dr. Joseph J. Skarypan has just received his commission as 1st Lt. in the Army Air Corps. We wish him luck in his future work.

1940—Lt. O. J. Giovanelli of the Army Medical Corps, is now stationed at Carlisle Barracks, Pa. He informs us that he will proceed to Hoff General Hospital in Santa Barbara, California, very shortly.

1941—First Lieutenant Joseph J. Nebrensky of the United States Medical Corps, has recently been promoted to the rank of captain. Captain Nebrensky is stationed at Daniel Field, Air Service Base Command in Augusta, Georgia. While in the service he studied at the Medical Field Service School at Carlisle, Pennsylvania, and at the Medical Service Training School in Warner Robins, Georgia.

1942—Lt. John R. Krolikowski, of the U. S. Army Medical Corps, is stationed at the Fourth Auxiliary Surgical Group at Lawson General Hospital in Atlanta, Georgia.

Dr. Lawrence Mazur, formerly of Chicago, is now a lieutenant in the Medical Corps.

"Enjoying the Quarterly," writes Dr. Gil Ehlers. Dr. Ehlers is engaged in general practice in Brookfield, Illinois.

1943—Our congratulations and best wishes to Dr. and Mrs. M. Dee Shapiro on the birth of a baby daughter, Linda Joy. Dr. Shapiro and his wife are residing in Brooklyn, New York.

FROM THE DEAN'S CALENDAR

Class of

1918—Major A. L. Schneider has recently returned from a two-year assignment in Alaska. He is now surgeon of the 1538 Service Unit, Camp Breckenridge, Kentucky.

1932—Lieut. Herman Rhoad has left Carlisle Barracks, Penna. to go to Fitzsimmons General Hospital, Denver, Colo.

1933—Dr. and Mrs. George Malcolm McClure of 348 S. Market Street, Paxton, Illinois called. Dr. McClure is in general practice.

1938—Capt. Herman R. Moser has visited the school frequently since obtaining his commission.

1938—Lieut. I. H. Blumenfeld has been doing service on a hospital ship.

1939—Capt. H. Tashman visited the school recently.

1939—Capt. F. C. Lawler has been stationed in the Canal Zone. Dr. Lawler left behind a prosperous practice in Chicago.

1940—Captain Ralph Wexler is Post Surgeon at Mara Loma Quartermaster Depot, in Mara Loma, California.

1941—Lt. Oscar Simon is stationed at Lovell general Hospital, Fort Devans, Massachusetts.

1941—Lieut. Frank Sabatino stopped in Chicago on his way to an Evacuation Hospital in California.

1941—Major M. J. D'Andrea visited the school recently

1942—Lieut. Joseph Palumbo stopped in on his way to Carlisle Barracks, Penna.

1942—Lieut. Allen W. Jackman called on his way to Carlisle Barracks, Penna.; from there he went to Tilton General Hospital, Fort Dix, New Jersey.

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We have learned that the following alumni have received commissions in the Army in addition to those already announced in previous issues:

1938—Lieut. Mario A. Accinno
1942—Lieut. Sidney Alpert
1941—Lieut. Morris D. Bennin
1928—Lieut. Anna C. Besick
1938—Lieut. Louis C. Curoso
1942—Lieut. Hansel M. DeBartolo
1940—Lieut. Ovidio J. Giovanelli
1942—Lieut. Walter I. Greenson
1942—Lieut. Irwin L. Greenspon
1942—Lieut. Allen W. Jackson
1938—Lieut. John Jacob
1942—Lieut. Lawrence Mazur
1934—Lieut.-Colonel John T. Porter
1943—Lieut. Robert D. Redston
1932—Lieut. Herman Rhoad
1942—Lieut. Abraham Schwartz
1941—Lieut. Oscar M. Simon
1938—Lieut. Joseph J. Skarypan
1942—Lieut. Samuel Stymacin

We are pleased to announce that the following graduates from the School have recently received promotions in the Army:

1932—Capt. Sidney R. Bazell
1934—Major Thomas H. Culhane
1941—Major Maurice J. D'Andrea
1940—Capt. Martin W. Green
1933—Capt. Abraham Kushner
1939—Capt. Frank C. Lawler
1941—Capt. Joseph J. Nebrensky
1931—Capt. Samuel B. Nuzie
1932—Capt. Charles Pava
1918—Colonel Edward H. Seifert
1941—Capt. Leo Tann
1937—Capt. Anton W. Wellstein
1939—Capt. Albert Wendorf
1934—Capt. Walter F. Zurawski

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Social Notes

We are pleased to announce the following weddings in the Junior Class:

Arthur B. Sincoff to Felice Siegel of New York, in New York on June 25th.

Marvin Rodney to Muriel Levy of Brooklyn in Brooklyn on June 20th.

Lawrence Weiss to Gertrude Rosenstock of New York in New York on June 18th.

Erik Larsen to Elsie Stemplel of Chicago in Chicago on June 17th.

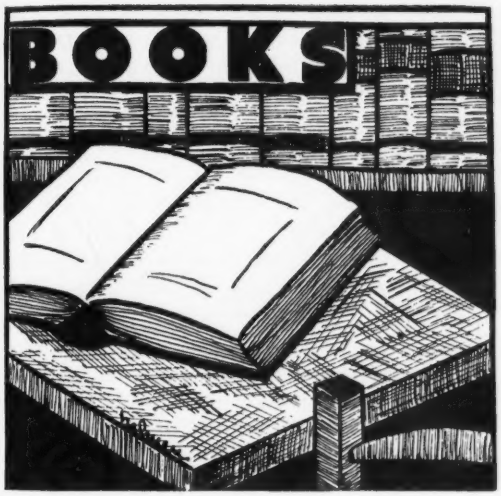
* * * *

Mr. and Mrs. Herbert Gornstein, class of '44, announce the birth of a son, Jon Creighton, at the Presbyterian Hospital, Chicago on March 30.

Mr. and Mrs. Leonard Tilkin, class of '44 (Ye olde Associate Ed) proudly announce the arrival of an 8 lb. son, Jeffrey Mitchell, at the Norwegian American Hospital, Chicago on May 3.

* * * *

The only way to get rid of a temptation is to yield to it.—Oscar Wilde.



FUNDAMENTALS OF IMMUNOLOGY, by William C. Boyd, Ph.D. xiv and 446 pages, 45 illustrations. Interscience Publishers, Inc. 1943, New York.

This book is more than an introduction of immunology. It offers not only the "fundamentals" of this science but a well-presented and thoroughly elaborated text for the study of all essential aspects of the subject. It is easy and pleasant to read, in spite of the liberal use of chemical and mathematical formulae. While much space is devoted to the discussion of the chemical and physical principles, one would like to read more about the application of immunological methods to bacteriology and parasitology. As a whole, Boyd's book is to be recommended to medical students.

DR. OSCAR FELSENFELD

LABORATORY METHODS OF THE UNITED STATES ARMY, edited by J. S. Simmons and G. J. Gentzkow. 823 pages, 103 illustrations, 8 color plates. Fifth edition. Lea and Febiger, 1944, Philadelphia.

Nothing is more difficult for a reviewer than to evaluate a handbook the former editions of which form an essential part of his library. It was a real satisfaction to see the "little brown book" grow up into a considerable, excellently edited volume, full of good illustrations and brief but useful descriptions of laboratory methods. The book reflects also the high scientific standard of the U.S.M.C. and U.S.Sn.C. Some chapters, e.g., those on virus diseases, Rickettsial infections, organisms of uncertain classification, etc. are more than "up to date." The parts treating bacteriology and parasitology are written in accordance with the present day status of science. Unfortunately little attention is paid to the typing of Salmonella, new media for V. cholerae and P. Pestis. These shortcomings, however, do not diminish the great value of the volume. (Who ever saw a perfect laboratory manual that covers the needs of all specialists?) It is the firm conviction of the reviewer that this book should occupy an important place in the library of every physician, scientist, and technician.

DR. OSCAR FELSENFELD

CLINICAL TROPICAL MEDICINE, by 27 authors, edited by Z. Taylor Bercovitz, xvii and 957 pages, Paul B. Hoeber, Inc., 1943, New York.

Twenty-seven authors, with the aid of an advisory board, compiled a considerable volume, each of them giving his own special experience in tropical diseases. Some of the chapters are fascinating—as those on dysentery, malaria, relapsing fevers, yellow fever, epidemic encephalitis, brucellosis, beri-beri etc. But one feels the lack of the description of cutaneous diseases other than those caused by yeast and fungi, the absence of a chapter discussing the course of cosmopolitan diseases in the tropics, tropical surgery, etc. The illustrations are very good; unfortunately there are few originals.

The material is well presented and the description of the pathology, ecology, symptomatology and treatment of most diseases is very good. Those who like to read a volume written by several workers, will enjoy the book very much. Those who prefer to study the opinion of one author, will remain faithful to Manson.

DR. OSCAR FELSENFELD

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HYPERTENSION

(Continued from page 13)

data (Tables 1 and 2) show that there is a relation between the reduction in plasma flow and the severity of the disease. The eye grounds were more severely damaged in the patients of the first group than in those of the second, the arteriolar wall was thicker, the vasomotor reactions slower. Seven of the 12 patients in the first group had malignant hypertension, 4 died and 1 probably died shortly after resection of the splanchnic nerves. Only in two cases did the blood pressure decrease permanently after the operation. On the other hand none of the patients of the second group had malignant hypertension; none died following the operation and in 6 the blood pressure was permanently reduced.

5. Examination of the vascular system. a) Determination of the blood pressure. The determination of the blood pressure is performed according to the recommendations of the American Heart Association[7]. The patient's pressure is measured in the sitting position on admittance to the hospital and every three hours until bedtime for three consecutive days. The determinations are made on both arms and the average of all determinations made is considered the patient's blood pressure before therapy. The blood pressure is arbitrarily considered normal if not above 130/90 in individuals under 40 years of age and 150/100 in individuals older than 40. Following an interval of at least six months after the beginning of therapy, the blood pressure is again determined, at least three times in each arm, according to the same technique. The blood pressure is considered "returned to normal" if it has fallen within the above defined limits. "greatly reduced" if there was a drop of at least 80 mm. systolic and 25 diastolic; reduced" if the reduction was of 40 and 15 mm. respectively; "increased' if there was a rise of at least 10 mm. systolic and 5 mm. diastolic; "unchanged' in all other cases.

b) Study of the vasomotor reactions. The vasomotor reactions are studied to determine to what extent the arteriolar lesions which are responsible for the increased peripheral resistance have become irreversible. Among the many methods suggested for this purpose, the following are relatively easy; the determination of the drop in blood pressure after 24 hours of bed rest, or after 30 minutes of abdominal diathermy; the determination of the rise in blood pressure after immersion of one hand in cold water (the so-called cold pressor test of Hines and Brown,[12] or during rapid mental arithmetic or after the breath is held for 30 to 60 seconds, or after standard physical exercise. Another method consists in the determina-

TABLE I

AVERAGE EFFECTIVE RENAL BLOOD FLOW, ETC. IN SEVEN NORMAL SUBJECTS AND IN 22 HYPERTENSIVE PATIENTS BEFORE OPERATION

	7 Normal Individuals	*Aver. Group I* Markedly Reduced Renal Blood Flow	*Aver. Group II* Moderately Reduced Renal Blood Flow	Average of Group I and Group II
Effective Renal Blood Flow (cc/min.)	1060.0	342.3	747.1	535.3
Glomerular Filtration Rate (CIn) (cc plasma/min.)	116.9	80.9	114.6	97.5
Filtration Fraction (CIn/CD) %	18.18	38.4	23.4	31.5
Blood Pressure:				
On Admission	.....	231/143	211/136	228/140
On Bed Rest	.....	198/123	150/57	181/109
During Clearance Test	128/76	205/130	173/111	190/120
Diathermy, Drop	.....	16/12	27/11	21/11
Cold Pressor Test, Rise	.....	21/18	38/24	29/21
Excretory Function Tests:				
Urine Concentration	.....	1.024	1.032	1.027
Urea Clearance	.....	75.0	107.04	89.7
NPN	.....	36.5	28.6	32.9
Arterioles Wall/lumen:	.....	1.241	0.9180	1.094
Keith-Wagner Classification of Eyegrounds				
No. of Patients: Normal	.....	0	3	3
I	.....	0	0	0
II	.....	0	1	1
III	.....	5	6	11
IV	.....	7	0	7

tion of the blood flow in the limbs, by means of a plethysmograph, following physical or pharmacological stimuli. The vasomotor reactions are more pronounced in patients with higher renal blood flow. These cases have better prognoses and better therapeutic results.

c) The direct observation of the blood vessels. This is possible in one of the following ways: 1) determination of the number and motility of the capillaries of the forearm following the method of Griffith.[13] This method is not easy and it is time consuming. 2) observation of the vessels of the eye grounds and classification of the patients according to the four following groups of Wagener and Keith[14]: First group, normal fundus; second group, sclerosis of the arterioles; third group, angiospasm; fourth group, papilledema. The importance of the examination of the eyegrounds is generally recognized as probably the best single method for the diagnosis and prognosis of hypertension. The statistics of Wagener and Keith demonstrate that the mortality increases from the first to the fourth group. From 5 to 9 years after the first physical examination the mortality was as follows: First group, 40%; second, 65%; third, 92%; fourth 99%. 3) Examination of the arterioles of the kidney and of skeletal muscles in biopsy material. Renal biopsy was recommended by Gerbi in 1938[15] and performed by Talbot and collaborators[16] in a limited number of patients. Before this method can be generally accepted it must be shown that it can furnish valuable information not otherwise obtainable, as it involves an operation which is by no means simple. The work of Fishberg[17], Moritz and Oldt[18], Wagener and Keith[14] and of others has shown that arterial hypertension is almost always accompanied by hypertrophy of the arterioles. To determine the degree of thickness of the arteriolar wall, samples of muscle, obtained by biopsy, are cooled to room temperature and fixed in formalin. During this procedure any vascular spasm disappears and only the permanent

(Continued on page 38)

PSYCHOTHERAPY

(Continued from page 9)

The fact that personal objections to the therapist are often mere rationalizations does not justify us, of course, in excluding the possibility of a mistake on our part. On the contrary, since we are all only imperfect human beings, all of us quite frequently make mistakes and commit errors. In most cases this does not do much harm. For everywhere in life mistakes can be remedied when the right understanding is present. And certainly our mistakes do not justify the patient in withdrawing from his obligations in life, or to put it concretely, in refusing to continue the course of the treatment. Through the revelation of these very interconnections and an open confession of one's imperfection, much may be accomplished toward the establishment of a really human contact between therapist and patient. If a patient nevertheless finds us objectionable because of our imperfections, he would probably do so even if we spoke with the tongues of angels or had other supernatural powers at our command. For in this case he is not yet ready to add anything himself to the improvement of his life-situation, that is, to change himself.

This combination of revolt against the therapist and flight from health often takes place because—as Adler emphasizes—rebellion plays a great role in any patient's life. This rebellion has not been provoked necessarily by the personal attitude of the therapist. The mere fact that the therapist appears as the representative of the human community, as the herald of the needs of human fellowship, will call forth that spite which the patient feels against human society. Up to now he has known how to withdraw successfully from life's demands and he will attempt to continue in this way if these demands are presented through the mouth of the therapist. This rebellion and the intention to escape from our interpretations probably make up the main roots of the resistance which occasionally occur in every treatment.

Though we have to reject the theory of a libidinous attitude on the part of the patient when we consider the diffculties of treatment, the question presents itself as to what role is played by impulses of love in the attainment of success in treatment. It probably infrequently happens that female patients are in love with their male therapist or that male patients fall in love with women therapists. This, however, seems not to be due to the character of the treatment as such. We must remember that all neurotic persons have special difficulties in love, because love is incompatible with the maintenance of special safeguards for one's self; and happiness in love demands the capacity for unconditional devotion. Discouraged, neurotic patients are not capable of this. Here, in relation to the therapist, they are in an unusually favorable situation. Nothing is demanded of them; on the contrary, the therapist attempts to get close to them, to understand them. Is it any wonder that the therapist may be idealized by the patient? Yet this

is not so common as one might think and as psychoanalysts are eager to maintain. Of course, some sort of tie between therapist and patient must exist. It could not be otherwise, considering the close personal cooperation and the measure of confidence which this work demands. The frequent discussions of most intimate problems, too, may perhaps occasionally call forth amorous impulses. However, the latter occur so frequently and easily in the life of every individual, unless he guards himself against them, that they cannot be considered as evidence of special love for the therapist. The desire not so uncommon among patients for entering into personal contact with the therapist also usually originates in their resentment at being nothing more than a case to him. They suffer from not being pampered enough, as they had always desired throughout life. They do not care to realize that before and after them there are other patients whom the therapist will meet with the same friendliness and courtesy. They want to be exceptions. Their interest in the therapist as a person originates in their striving for prestige. Perhaps occasionally some women patients will offer the only treasure which alone in their feeling of inferiority they believe they still possess: the fact that they are females. But even this does not justify us in supposing that any real attachment for the therapist exists.

Since the hypothesis of love for the therapist is a dubious one, this applies even more in estimating the effect of this factor in relation to the success of the treatment. Occasionally a special affection for the therapist will increase the readiness to accept his point of view. It is, however, more than doubtful whether such an acceptance of insight really signifies a lasting success. For when this insight is built upon a feeling of love for the therapist, the logic of reality sooner or later will bring disappointment. And with this the entire ideological superstructure is likely to collapse. Confidence and newly-won courage are followed then by an even deeper depression. It may also happen that a woman patient, feeling that the psychiatrist may—to use the characteristic phrase—become "dangerous" to her, will be tempted to abandon the treatment. In other cases, too, affection for the therapist may tend to impede the treatment, even when this feeling is "brought to consciousness and is not found objectionable." How often is the treatment nullified under these circumstances! The therapist as a man is placed on a pedestal so that whatever he says in his professional capacity may be discounted. The content of his arguments is dismissed as negligible, while the patient regards only his personality as important. This apparently positive attitude is very often aimed directly against the treatment.

Even without "love," a certain degree of mutual experience in the treatment is possible, yes, even very important for its progress. The patient in the doctor's office shows himself in his true colors. He behaves no differently than he does in his outside life. And his attitude toward the psychiatrist reveals the sum total of his personality as does the review of his past life, his characteristic experience, his dreams, etc. When the patient learns to understand this, he will know himself. And if he behaves well in the treatment, in this definite task, then he will also behave well in life.

The characteristics and the life-style of a patient are revealed with emphatic clarity in the course of collective therapy. In the joint discussions with several patients, the latter learn to know themselves with much more ease because they are able to make observations about one another. Once the first resistance against taking part in an association for the purpose of therapeutic treatment has been overcome, much ground has been gained on the way to a quicker and more thorough recognition by the patient of his own personality. Above all, this method eliminates any last remnant of a personal battle with the therapist. For as long as psychiatrist and patient face one another, in any sense of competition, the latter will feel himself threatened in his prestige. He may regard his possible cure as a personal defeat and may consider the treatment's failure as a defeat for the therapist. Conduction of the treatment in the spirit of a duel is the greatest handicap to its successful outcome.

It is therefore important that any authority, yes, even any comparison of respective personal values, should be avoided during the treatment. The patient, who shows his life-style in his behavior during his treatment, attempts to measure his strength against the therapist's and to construct an "above" and "below," instead of a cooperation of equals. Either he directly repudiates the therapist from the fear that otherwise he might feel his own inferiority too strongly, or he places the therapist in a special position to him. In this case he believes in the therapist's "power of suggestion" or he expects that this be brought into play, sometimes demanding it directly and explicitly. It may be that the role which the therapist is supposed to play is, as Freud believes, often patterned after the image of the father. If so,

it is only because the patient had learned as a child to misuse the dreaded authority of his father as an excuse for his own weakness. It is much more comforting for the patient to acknowledge the therapist's authority. Thus he is relieved of any obligation to work toward his cure himself. And, above all, this procedure burdens the psychiatrist with full responsibility for success and failure of the therapy.

Hence it is imperative to reject consistently the attempt of the patient—which is made in the course of every treatment—to represent himself as small and dependent, the therapist, on the other hand, as the omnipotent leader. For many a patient it is no doubt a heavy burden to have to realize that it is not the therapist and not any mystic "treatment" that is to make him well, but that he himself alone is able to do this. For then he is oppressed by his pondering over the question of why he did not take this path long before, if he alone is the one upon whom it devolves. Some miracle performed by the therapist ought, he feels, to free him of the mistakes of his past. Nevertheless, the recognition of the patient's own strength is the foundation for a cure and for the permanence of its success.

For this reason prescriptions which are given for the treatment of psychoneuroses—often with the best intentions—can be harmful. Medications as well as prescribed rules of behavior do not strengthen the patient's self-confidence, which alone can help him in solving the problem of himself and his own life, of his relationship to others. Prescriptions rather infringe upon it. What is most important in every treatment is encouragement. The most effective therapeutic factor is shown to be the uncovering of unjustified feelings of inferiority. On the other hand the revelation of the striving for prestige often calls forth obstinancy, for the neurotic striving for prestige disappears only when the feeling of inferiority, which is its basis, is overcome. Revelation and challenge of the accustomed life-plan, and encouragement: these are decisive factors with which our treatment is concerned. Only in that way can the social interest of the patient be increased, his social adjustment, which is synonymous with his cure, be accomplished.

What do the robins eat, Mummy?
Worms, dear.
And what do the worms eat?
The dead robins, darling.

—*Stephen Leacock*

DIABETES

(Continued from page 19)

Keep patient on *insulin-HCl* for at least *3 days,* preferably 4 or 5 days.

3. Protamin-Zn-Insulin: After patient has been on insulin-HCl for at least 3 days, give insulin subcutaneously once a day after breakfast as follows;

Protamin-Zn-Insulin 2/3 of total insulin dosage, e.g. 50 units.

Insulin-HCl 1/3 of total insulin dosage, e.g. 20 units. Don't mix two types of insulin because better results are obtained if each is given in two separate sites. (A true diabetic cannot be managed well on Protamin-Zn-Insulin alone).

Protamin-Zn-Insulin takes care of "night blood sugar," while insulin-HCl takes care of "day blood sugar":

	Onset	*peak*	*end*
action of			
Protamin-Zn-Insulin	12 hrs.	18 hrs.	24 hrs.
action of			
Insulin-HCl	½ hr.	4 hrs.	6 hrs.

4. Watch urine for sugar and acetone. A slight glycosuria (plus 1 or 2) is of no consequence.

As patient's condition improves, his carbohydrate tolerance usually increases, and the amount of insulin given may be reduced. In the presence of infection, it is a wise precaution to increase the insulin dosage and carbohydrate intake. After the patient is well-regulated, instruct him to run his urine specimens 3 times a week (fasting specimens), and to report his progress to the physician every two to four weeks.

MEDICINE FOR THE HEART

The late Dr. Luther Emmet Holt, the great baby specialist, had a standard treatment for all frail newborn babies who failed to gain weight. When he came to the chart of such an infant during his hospital rounds, he always wrote the following direction for the nurses:

"This baby to be loved every three hours."

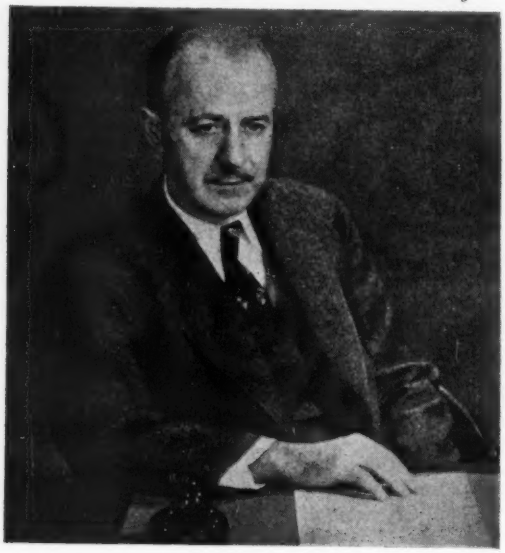
* * * *

A young lawyer, pleading his first case, had been retained by a farmer to prosecute a railway company for killing 24 hogs. He wanted to impress the jury with the magnitude of the injury.

"Twenty-four hogs, gentlemen! Twenty-four! Twice the number there are in the jury box."

Faculty Notes

Dr. Andrew Howard Ryan has recently been appointed Associate Professor of Physiology and Pharmacology at The Chicago Medical School. Dr. Ryan brings with him a most varied and extensive background in the physiological sciences.



Dr. Ryan received his M.D. degree from Washington University in St. Louis. He was Instructor in Physiology and Pharmacology at the University of Pittsburgh, and held the chair of Physiology at the University of Alabama, Tufts Medical School, and the University of Maryland. He was Lecturer in Applied Physiology at Yale University, where he was the originator of the subject which has since been carried on successfully. Dr. Ryan was also an Associate Physician with the United States Public Health Service.

Dr. Ryan has carried out research in many fields, including asphyxia and resuscitation, goiter, action of magnesium sulfate, caffeine, and strychnine. He is at the present time engaged in a series of experimental studies of fatigue in Man, including quantitative measurements of fatigue and the relation of fatigue to efficiency in industry. He has recently had papers published in the American Journal of Physiology, the Journal of Pharmacology and Experimental Therapeutics, and the Journal of Industrial Hygiene.

Besides being a member of Alpha Omega Alpha and Nu Sigma Nu, Dr. Ryan holds membership in the American Physiological Society, the Society of Pharmacology and Experimental Therapeutics, the American Statistical Association, and the National Institute for Industrial Psychology in Britain.

Dr. Ryan is married and has three children. He likes to play chess and contract bridge, enjoys tennis and handball.

We are happy to welcome a man of Dr. Ryan's experience and ability to The Chicago Medical School.

Hereafter, under Faculty Notes, an effort will be made to keep the reader informed of recent publications of, talks given by, and honors bestowed upon members of the faculty.

Dr. J. N. Essenberg.

1. "Signs, Scientists, and the Scientific Method." Delivered before the Latvian Club, March 19, 1944.
2. Essenberg, J. N., & Momigliano, E. March, 1944. Regenerative Processes Induced by Gonadotropic Hormones in Irradiated Testes of the Albino Rat. Radiology, vol. 42, pp. 273-282.

Dr. O. Felsenfeld.

1. Felsenfeld, O. 1944. Yeast-Like Fungi in the Intestinal Tract of Chronically Institutionalized Patients. Amer. J. Med. Sci., vol. 207, pp. 60-63.
2. Felsenfeld, O., & Young, V. M. 1944. The Occurrence of Members of the Genus Salmonella in Inhabitants of State Hospitals of the Greater Chicago Area. J. Lab. and Clin. Med., vol. 29, pp. 375-382.
3. Felsenfeld, O., & Young, V. M. 1944. A New Medium for the Differentiation of *Salmonella* and *Paracolon* Organisms. American Journal of Clin. Pathology, vol. 14, pp. 26-27.
4. Felsenfeld, O. 1943. Summary of Technic for Identification of Organisms of the Typhoid, Paratyphoid, and Dysentery Groups. *Clinical Laboratory Methods and Diagnosis*. R. B. H. Gradwohl, 3d Ed. C. V. Mosby Co., St. Louis. Vol. II, pp. 1203-1227.
5. Felsenfeld, O., & Young, V. M. 1944. The Immunological Aspects of Vaccination Against Cholera with Bacterial Antigens and Toxoids. Paper prepared for the Annual Meeting of the Society of Tropical Medicine.
6. Felsenfeld, O. 1944. Selective Media for the Isolation of *E. typhosa* and *Salmonella* for a Hot Climate. Annual Meeting of the Society of American Bacteriologist, May 6-8, 1944.
7. Felsenfeld, O. 1944. Bacillary Dysentery (Diagnostic Media). Med. J. of Australia, vol. 31 (I), p. 137.

Besides the above activities, and others not listed, Dr. Felsenfeld has been honored by being elected to membership in the American Microscopical Society, and to Fellowship in the Royal Society of Tropical Medicine and Hygiene, London.

Dr. P. P. Foa.

1. "Transmethylation." Delivered before the U. of Illinois, Physiology Journal Club, May 22, 1944.

Dr. V. Levine.

1. "Intestinal Histoplasmosis." Delivered before the Chicago Pathological Society, January, 1944.

Dr. W. E. MacFarland.

1. MacFarland, W. E. April, 1944. Adrenal Cortical Function Independent of Direct Nervous Action. J. Exp. Zool., v. 95, pp. 345-355.

Dr. I. S. Nieman.

1. "The Chronology of Allergy and Immunity in Tuberculosis." Read before a meeting of the American Academy of Tuberculosis Physicians, June 12, 1944.

Dr. R. G. Roberts.

1. "Spectroscopy of Proteins, Free and in Situ." Delivered before the University of Illinois, Physiology Journal Club, May 3, 1943.
2. "Physical and Chemical Properties of Macromacules." Delivered before the University of Illinois, Physiology Journal Club, Dec. 20, 1943.
3. "Inclusion Bodies and Viruses, Elementary Bodies and Proteins." Delivered before the University of Illinois, Physiology Journal Club, June 5, 1944.
4. Dr. Roberts has also applied for a patent on Insulin Ferrihemochromogen.

Dr. J. A. Smith.

1. "Oxygen." Delivered before the University of Illinois, Physiology Journal Club, March 15, 1943.
2. "Physiology of Salicylates." Delivered before the University of Illinois, Physiology Journal Club, Feb. 14, 1944.
3. "Relation of Vit. E to Metabolism." Delivered before the University of Illinois, Physiology Journal Club, May 29, 1944.

Miss V. M. Young.

Miss Young has been junior author of many articles written with Dr. O. Felsenfeld which are included above. She has also accomplished the following:

1. "Salmonella Strains in Mentally Deficient Children." Annual Meeting of the Society of American Bacteriologists, May 6-8, 1944.
2. Young, V. M. & Felsenfeld, O. 1944. The Incidence of *Endomonas intestinalis* Wenyon and O'Connor in Food Handlers and Diarrheic Patients of Mental Hospitals. J. Parasitology, vol. 30, pp. 34-35.

Miss Young was also honored by being elected to membership in the American Microscopical Society.

Organizations

The Chicago Medical School Chapter of the A.I.M.S. continued its film series the past quarter and made plans for a still further expansion of its functions in the coming months. The plans include the usual series of interesting and educational films, social affairs, prominent speakers, war services, and an A.I.M.S. *News-Letter*.

The films to be shown soon include "Physical Examination of the Infant," "Tuberculosis in Childhood," "Pediatric Anomalies," "Allergy," "Pneumonia," "Orthopedic Treatment of Infantile Paralysis," and "Technique of Blood Transfusion." A Faculty-Student Dance is being planned. The War Service Committee is making arrangements for a mass excursion to the Red Cross Blood Donor Center.

At the recent elections, Emanuel Chusid was elected president; Harold March, vice-president; Arnold Statsinger, treasurer; and Ruth Steinman, secretary. The following committees were appointed: social committee, Mel Morrel, chairman; program committee, Dominic Artuso, chairman; and publicity committee, Bernard Shulman, chairman.

With the 100% enrollment of the freshman class, the Chicago Medical School Chapter of the AIMS has an excellent opportunity to extend its advantages to the great majority of the student body. Under its new leadership, a rejuvenation of activity can be expected, with benefits to the school and student body alike.

PHI LAMBDA KAPPA

The Alpha Rho chapter of the Phi Lambda Kappa fraternity pledged twenty-eight prospective members at a recent dinner meeting. A pleasant innovation at this affair was the presence of female guests.

The guest speaker of the evening was Dr. Sicher of Loyola University who presented an enlightening and forceful talk on racial prejudice. After pointing out the fallacy of racial superiority from an anthropological viewpoint, he showed how one group, through social pressures, can perpetuate in another group those characteristics upon which the first group claims to base its prejudicial attitudes. Dr. Sicher's solution for breaking this vicious cycle is that the members of the second group must individually so conduct their lives as to refute the claims of the first group, at the same time not exhibiting similar attitudes toward other groups.

Among the guests present were Dr. and Mrs. Samuel Taub, Dr. Loeffler, and the Honorary Worthy Superior, Dr. Brodsky.

On Decoration Day, some of the members and their guests attended a picnic held by the Alpha Alpha chapter. The occasion was a success, and similar affairs are planned.

* * * *

A skilled lens grinder, Baruch Spinoza, was frail and tuberculous, but his philosophy has had an enormous influence upon modern thought. Breathing glass dust by day, pouring nightly over his books, always closely confined, dieting in the hope of curing his illness, the constitution of Spinoza was soon worn down as surely and completely as the lens he ground by day. Death came before anyone realized how seriously ill he had been.

Letters To the Editor

Dear Editor:

I was in the class of 1936. Would appreciate getting the QUARTERLY out here. I am with the Amphibious Engineers who have made landings and invasions all the way from Africa to Sicily to Italy.

At present we are sweating it out on the Anzio Beachhead. I have a small underground hospital, and am getting along fine. Out here the whistling of shells and the enemy air raids keep things from getting too monotonous. We are doing a good job, and expect to keep doing so until this thing is over. Regards.

Capt. Bernard L. Cohen, M.C.
1st B'n, 540 Eng'r.
A.P.O. No. 464 c/o P.M. New York City

(Thank you for your address, Captain. We will see that you get your copy of the Quarterly henceforth.)

To the Editor:

The Student Council is formed. This is solely a response to a need which has often been stated in these pages and elsewhere. It would surely have occurred sooner or later. That it has occurred sooner is due to the efforts of the following members of the Committee For A Student Council and to the support given us by the student body:

Leonard Arnold	Burton Rockcliff
Lester Wolff	Robert Bodwin
Louis Richman	Milton Wohl
Marvin Loring	Sally Major
Henry Schnitman	William Shlaes

Special recognition should be given the Quarterly whose editorialist, in the December 1943 issue, presented a challenge which could be answered only in the formation of a Council.

The Council was elected by the student-body. Its Constitution was ratified by the student-body. Whatever it may achieve will depend upon the active support of the student-body.

Respectfully,
A. J. Zweibel, Chairman,
Committee For A Student Council

* * * *

Excerpts from the *Guide for Physicians* by Isaac Judaeus.

"The Physician does not make the cure. He merely prepares and clears the way for Nature, who is the real healer."

WATER METABOLISM

(Continued from page 6)

above. It varies, too, with the humidity, for the faster the evaporation the greater the cooling and the less need for perspiration. According to Zunz, a soldier using up 1000 calories on a march at 10° C. in saturated calm air loses 800 gms. of water. Each 1° C. increase in temperature raised this water loss by 38 gms; each unit of wind velocity increase lessened it by 10 gms. Work in hot climates may produce enormous water and salt losses and is responsible for heat stroke and heat cramps. The relationship between the loss of water and that of salt will be discussed later.

Under ordinary circumstances, the loss of water from the alimentary canal is negligibly small in spite of the magnitude of the digestive secretions. Gamble gives the following approximate values for the 24 hour digestive secretions:

Saliva	1500 cc.
Gastric secretions	2500 cc.
Bile	500 cc.
Pancreatic Juice	700 cc.
Intestinal Juices	3000 cc.
Total	8200 cc.

This startling total of more than 8000 cc. is almost completely absorbed by the time the fecal mass reaches the anus.

Peters has drawn the following conclusions from modern studies of alimentary secretions: "First, despite the diversity of their chemical patterns, the digestive secretions are, with the possible exception of saliva, isotonic with blood serum. (Saliva appears to be somewhat hypotonic). Second, mixtures introduced into the stomach or gut become isotonic with serum in the course of their absorption. Third, most of the fluid poured into the intestine from the body is subsequently reabsorbed. Fourth, much of the material of which feces is composed represents products contributed to the gut in the gastrointestinal secretions and is not merely unabsorbed ingesta."

In diseases of the gastrointestinal tract profound disturbances in water balance may be produced by abnormal losses of alimentary secretions and by the lack of ingestion or absorption of water from beverages and food. Such a disturbance can occur during protracted vomiting from any cause but particularly in pyloric or intestinal obstruction. It may also occur in severe dysenteries such as are seen in diarrhea of

infancy, bacillary dysentery, and in cholera. A more detailed picture of such disturbances will be given later.

It is in the kidney that the refinements of water balance are executed. In fact, the function of the kidney can be defined as the maintenance of the constancy of volume and composition of the interior environment of the animal organism. The evolutionary development of the kidney has been influenced chiefly by the vicissitudes in the availability of water and in its osmotic content in the external environment of animals. Parallel to the changes in the kidney to meet these variations have been changes in the disposition of ammonia formed from the deamination of protein amino acids. In most aquatic organisms, with the exception of the marine elasmobranches, the major nitrogenous end product is ammonia, for this substance, though toxic in concentrated form, can be eliminated rapidly in dilute solutions by these animals, since there is no limitation in the water supply. In the amphibia and higher mammals, the toxic ammonia is converted to the relatively innocuous urea, so that it can be eliminated safely under conditions of limited water intake. However, to excrete urea, an appreciable quantity of water must be lost even when the supply is limited, for, as the urea solution becomes more and more concentrated in its passage down the renal tubules, its osmotic pressure finally becomes high enough to prevent further reabsorption of water by the tubular cells. Thus, an adult person has to excrete some 500 cc. of urine in order to eliminate the 22 gms. of urea formed in the average daily metabolism of protein.

Birds and reptiles have even greater limitations of water supply than do other terrestrial vertebrates, in that reproduction takes place in an egg surrounded by a relatively impermeable shell (the so-called cleidoic egg). Urea could not be used as the nitrogenous end product in such embryos, because the excretion of urea into the allantois would require an accompanying excretion of water great enough to deplete the organism. In such embryos, and also in the adults, water conservation is accomplished by converting the ammonia into uric acid which is excreted in dilute solution by the kidney but permits the almost complete resorption of water from the cloaca, since uric acid is so insoluble that it precipitates out and does not exert any osmotic hindrance to water resorption. The uric acid remains in the allantois as solid crystals; in the adult bird, it is excreted with the feces as a semisolid paste.

The recent researches of Homer Smith have demonstrated that to excrete 1 cc. of urine per minute, some 1200 cc. of blood or 650 cc. of plasma must pass through the kidney per minute. Of this 650 cc. of plasma, as it passes through the glomeruli, 20 per cent is filtered forming about 130 cc. of glomerular filtrate. From this 130 cc., 129 cc. of fluid are resorbed in the passage down the tubules to form 1 cc. of urine. This resorption takes place in two stages, an obligatory resorption involving about 90 per cent, and a facultative resorption of the remaining 10 per cent. The obligatory resorption is passive and is probably operated by the increased colloidal osmotic pressure of the unfiltered and therefore concentrated blood as it passes along the capillaries surrounding the tubules. The facultative resorption, which probably takes place in the ascending loop of Henle and the distal convoluted tubule, is an active process, and is undoubtedly influenced if not controlled by such hormones as that of the posterior pituitary and that of the adrenal cortex. It is in this facultative resorption, with its accompanying adjustment of urine acidity and base conservation that the real refinements of water balance are carried out.

III Cation-anion Balance of Serum and Interstitial Fluid.

In human blood plasma, since the pH is about 7.4 or practically neutral, the total concentration of cations is about equal to the concentration of the anions, if these are expressed in equivalent terms. Expressed as milliequivalents per liter, the normal concentration of the plasma electrolytes is exemplified by the following data.

Anions	m.eq./liter	Cations	m.eq. per liter
Chloride	104	Na	143
Phosphate	2	K	5
Protein	16	Mg	2
Sulfate	0.5	Ca	5
Organic Acids	5.5		—
Bicarbonate (HCO_3)	27	Total Base	155
	155.0		

The concentration of total base in the blood plasma is one of the most constant factors in the maintenance of the normal physiological state. It is a measure of the osmotic pressure of the body fluids. A variation of more than 4 per cent in either direction represents a pathological stage.

Of the total base, 27 milliequivalents per liter, or that amount per liter which is left over after the

neutralization of the non-volatile acids,—chloride, phosphate, sulfate, protein and organic acids,—is available for the neutralization of CO_2 that is constantly being formed and which must be neutralized before excretion. This is the alkali reserve. Under normal conditions, this remains about 27 m.eq. per liter or about 60 cc. of CO_2 per 100 cc. of plasma as determined by the Van Slyke CO_2—combining power estimation. When other acids, accumulate in the body, as for example in diabetes or in uremia, there is less base available for the formation of bicarbonate. The alkali reserve is thus decreased, and unless there is a corresponding reduction in the amount of free H_2CO_3 held in the blood by rapid deep breathing, an increased acidity of the blood occurs. In diabetes, it is the accumulation of acetoacetic and β-hydroxybutyric acids that is responsible for the acidosis; in uremia it is usually the retention of sulfate and phosphate that lowers the bicarbonate.

The constancy of the total base concentration is maintained chiefly by the constancy of the sodium concentration. Those conditions associated with a loss of sodium will produce a corresponding loss of water so that the concentration of the total base in the remaining plasma and other extra cellular body fluids remains normal. In diabetes and chronic Bright's disease, such a loss of sodium occurs and the concomitant loss of water also produces dehydration. When the dehydration is moderate, the total base concentration remains normal. However, with the continued loss of sodium in the urine, eventually the loss of water lags behind so that as the dehydration becomes more severe, there is a drop in the concentration of total base. In severe cases, it may go as low as 130 m.eq. per liter. In such a condition, not only is there less than the normal amount of extracellular fluid in the body, but each liter of remaining fluid has a lower osmotic pressure than normal and has a lower amount of base available for neutralization of acids. Usually in these cases, there has been a corresponding loss of chloride and thus there may be no real diminution in alkali reserve per liter. But if the chloride has not been correspondingly lowered, there will be a loss of alkali reserve from this uneven loss of base.

In both diabetic and uremic coma, the condition is not only that of uncompensated acidosis as above described, but there is also marked dehydration and the fluid remaining in the body is low in sodium and chloride. From the clinical point of view, treatment must be aimed, not only to counteract the cause of the condition, as giving insulin for diabetic coma, but also to restore the normal electrolyte picture. Hypodermoclysis or intravenous injection of physiological NaCl solution will accomplish the purpose, since it will restore water, sodium and chloride, and the excess of chloride will be excreted in the urine. In uremia such therapeutic measures are similarly necessary, with the danger that the patient's limited excretory ability may substitute a chloride acidosis for the previous one. This can be avoided by adding isotonic sodium lactate to the salt solution, that is, using Hartmann's solution.

In pyloric obstruction, the loss of water, HCl, and NaCl produces a picture of dehydration, low total base, excessively low chloride and a corresponding high alkali reserve, resulting in alkalosis and tetany. In such cases, hypodermoclysis of physiological salt solution can restore normal hydration and normal electrolyte balance, if the kidneys are able to excrete the excess sodium as $NaHCO_3$. In intestinal obstruction and in the diarrhea of infants, water, chloride, and an excess of sodium are lost, producing dehydration, low total base, and a relative chloride acidosis. The therapy is similar to that of other acidoses.

Examples of the electrolyte disturbance superimposed upon dehydration in these conditions are given in the following table:

	Normal	Diabetic Coma	Uremic Coma	Pyloric Obstruction	Intestinal Obstruction
Total Base	155	135	135	140	135
Cl	103	84	86	78	93
PO_4	2	2	7	3	2
SO_4	0.5	0.5	7	1	1
Protein	16	16.5	16	16	16
Orig. Acids	5.5	20.0	5	7	5
Bicarbonate*	28	12	15	35	18

There are conditions as have been previously mentioned, associated with the retention of water, sodium or chloride in the body, as for example in nephrotic type of edema. In such conditions, there is a lag in the return of fluid from the extra cellular spaces back in the blood stream because of the lowered osmotic pressure of the plasma produced by the low protein concentration. The ingestion here of either water, sodium or chloride in quantities greater than can be simultaneously excreted is associated with the retention of the other two constituents, so that, though edema results, the retained fluid as well as the rest of the extra cellular fluids of the body have a normal osmotic pressure as shown by a normal total base content. In diuresis and loss of edema fluid,

sodium, chloride, and water are parallelly lost, so that the remaining fluid of the body does not alter in total base concentration. In intractable cases of edema, the total base or sodium concentration of the plasma is often less than normal and less than the threshold value for the excretion of sodium. In such cases, diuresis usually cannot be obtained unless the sodium content of the serum is elevated.

Since each liter of extracellular fluid contains a constant quality of total base, part of which is reserved for neutralization of CO_2, then it follows, that, while dehydration itself means a lowering of total alkali reserve, the reverse is true in edema—there is an increase in the total alkali reserve.

From a practical clinical point of view an adequate picture of the water and electrolyte balance can be obtained by making determinations of serum sodium, chloride, and CO_2-combining power. If the serum sodium concentration is below the minimal normal of 138 m.eq. per liter, then severe dehydration is present. If the chloride concentration is correspondingly lowered, then any lowering of CO_2 combining power that may be present must be due to the accumulation of other acids, as mentioned previously. If chloride is relatively higher than sodium, replacement therapy should include, besides saline solution, extra alkali, either in the form of $NaHCO_3$ or sodium citrate, if these can be given by mouth; or as sodium lactate, if injected intravenously.

Though the injected saline solution may also contain glucose, the latter must not be given alone in water as a replacement therapy, when the serum sodium is lower than normal, for not only will the water not be retained, but when it is lost it will carry more sodium with it, and the patient will be more dehydrated than before.

Conclusion:

In this rapid survey of water metabolism, we have seen its importance in a variety of biological processes. Life began in the sea, and the imprint of that beginning is still visible. The cells of the highest mammals carry out their life processes in an environment comparable to those of the primitive aquatic organisms. But to do so, these higher animals have had to develop highly intricate mechanisms. It is not strange, therefore, that in disease processes, water metabolism and that of its accompanying electrolytes are so frequently and so violently disturbed. The investigators of the last twenty-five years, however, have helped us materially to understand these disturbances, and to apply this information to the restoration of normal water metabolism.

SURGICAL COMPLICATIONS

(Continued from page 16)

oxygen and CO_2 inhalations, encouraging the patient to take deep breaths and to cough, and frequently changing his position. Other measures might be necessary, such as aspiration through the bronchoscope of the mucous plug that is damming up the bronchus and placing the patient on the side opposite the involved lung. Diathermy to the chest is also of value.

Pneumonia:

Post-operatively, this is usually of the broncho-pneumonic type and is a true pulmonary infection. Many conditions thought to be broncho-pneumonias are in reality patchy atelectasis and frequently clear up. The same preventive measures mentioned under atelectasis help to avoid pneumonia which, when it occurs, requires such specific treatment as the proper type serum, sulfadiazine or sulfapyridine and oxygen therapy.

Pulmonary Embolism:

This is the most dreaded, most treacherous and most fatal of complications and is caused by a sudden blockage of a large pulmonary artery by a blood clot which comes from a large vein usually of the pelvis or lower extremity. Pulmonary infarction is the same except that it implies obstruction of a smaller pulmonary artery by a smaller clot. Infarction occurs more often than massive embolism and is mild in character and often goes unrecognized and is mistaken for a pleuritis, bronchitis, or mild pulmonary edema. As mentioned before, early post-operative hyper-ventilation, changing posture, and frequent leg exercises form the chief preventive measures.

When the dramatic picture of sudden severe chest pain, cyanosis, rapid pulse, cough productive of a bloody sputum, and dyspnoea occur, there is no question about the diagnosis. Death may occur suddenly or may be delayed for several days. Operative treatment, as advocated by Trendelenburg, is hardly ever performed in this country because of its high mortality and the questionable accuracy of diagnosis in the first place. Another objection is that it would have to be performed by the inexperienced resident, intern, or junior surgeon. Non-surgical treatment therefore offers the better hope, and should consist of administering morphine to allay pain and apprehension, oxygen inhalation for the anoxemia, papaverine gr. ½ and atropine gr. 1/200 intravenously, as advocated by DeTakats, so as to overcome the severe arterial spasm in the lung that always accompanies sudden thrombosis and embolism.

Thrombophlebitis and Phlebothrombosis:

Deep venous thrombosis usually occurs in the lower extremity and because it is a forerunner of pulmonary embolism is a most dreaded complication. Two forms of this occur: a) Thrombophlebitis or obstructive intra-vascular clotting due to inflammation of the wall of the vein forms a firm, attached clot. This is the harmless form and goes by such names as *Phlegmasia alba dolens* and *milk leg*. It is associated with pain, fever, and circumferential swelling of the leg. Because it is attached, it rarely causes pulmonary embolism. b) Phlebothrombosis or insidious non-obstructive intravascular clotting forms a soft and loosely attached clot that breaks off easily. This is the treacherous form that causes sudden fatal pulmonary embolism. With this type there is most often no warning, no swelling, no pain, and no fever.

Superficial venous thrombosis, such as thrombosis of the saphenous vein, is easily distinguished from deep thrombosis by the following signs: visible palpable thrombi and pain and tenderness over the superficial thrombosed vein; fever, and patchy swelling over the involved part of the extremity.

One of the earliest signs of deep vein thrombosis is deep pain in the extremity especially on dorsiflexion of the foot (Homan's sign) and a slight rise in temperature. The pain is usually in the calf of the leg and symptoms occur most frequently after the fifth day and as late as 35 days. Sharp pains occurring in the chest assciated with cough, dyspnoea, and bloody sputum speak for pulmonary infarction that may be followed by massive pulmonary embolism. On the other hand there may be a sudden fatal embolism with no clinical signs of previous deep vein involvement as is the case with phlebothrombosis. The fewer the symptoms in thrombophlebitis the greater is the danger of pulmonary embolism. Venograms made by injecting diodrast into the small sephenous vein at the ankle may be the only way of revealing the silent thrombus in the deep veins and being able to tell in what extremity it is located.

The treatment of thrombophlebitis consists of bed rest, elevation of the extremity, application of moderate heat to favor vasodilation and gentle movements to overcome muscle spasm. Because there is an accompanying arterial spasm that produces the severe pain, papaverine given in doses of ¼ to ½ gr. intravenously is effective in relieving this painful arterial spasm. More effective is an early paravertebral sympathetic nerve block using 1% novocaine solution and injecting 5 to 10 cc. at each of the 5 lumbar segments on the involved side. When this is used early, there is prompt relief of pain and swelling. If pain and swelling recur, the treatment is repeated daily as needed. Recovery is usually prompt and the sequelae of chronic edema such as induration, pigmentation, and subsequent ulceration are avoided. Small doses of deep X-ray therapy likewise may help to absorb the perivascular exudate or edematous fluid.

Anti-coagulation therapy, employing heparin intravenously should be started early in every case if it be suspected that pulmonary embolism may occur or that phlebothrombosis is present. The drug is given for at least 3 days in sufficient amount to keep the coagulation of blood at 15 minutes. Because of its expense it may be supplemented by oral treatment with dicoumarol. Since dicoumarol requires about three days to become effective, it should be started with the heparinization and then continued for at least 14 days. Ligation of the femoral or iliac veins is to be done only if the above treatment fails and there is evidence of lung infarction. The ligation is done best through an incision parallel to and just below Poupart's ligament as for a high saphenous vein ligation. The saphenous vein is followed to its insertion into the femoral vein. The deep femoral vein is retracted and two tapes are placed one inch apart around the superficial femoral vein. A slit is then made in the vein between the tapes and the clots evacuated or sucked out by aspiration until a free flow of blood is effected. The vein is then doubly ligated by transfixion with silk and is cut between the ligatures. If adherent clots are found and no free bleeding occurs, a dissection is made above Poupart's ligament (as for exposure of the ureter) and the iliac vein uncovered. The vein is then opened, clots evacuated, then ligated and severed between the ligatures. The resulting edema of the leg clears up in about 6 months provided the patient will wear an elastic compression bandage every day. Recently at many of the army hospitals there is a trend to perform promiscuous ligation of the femoral and iliac veins and even the vena cava for deep ilio-femoral thrombosis. I fear that such promiscuous ligation will take its toll later by producing some form of vascular disturbance in time to come.

The preventive treatment of thrombosis has always stressed careful technique—sharp dissection so as to keep trauma to a minimum and good hemostasis—correction of dehydration so as to keep the blood less viscid, avoidance of excessive heat loss at operation for this slows the circulation, and avoidance of tight

bandages and dressings, for these obstruct the blood flow. Deep breathing and frequent leg exercises, started immediately after operation, with frequent changes in posture, turning the patient from side to side, and elevating the buttocks and lower extremities, increases the venous blood flow and prevents stasis. If all of these precepts are followed, thrombosis will be infrequent.

Hepatic, Renal, and Cardiac Failures:

Despite every precaution, deaths will occur at times in the cirrhotic, nephritic, and cardiac patient who has been properly prepared. Disaster can usually be ascribed to a break in the cardiac compensation, or to a sudden suppression of kidney and liver function by toxic effects of the anesthesia, by blood loss, or by a sort of shock to the vital organs from the operative trauma.

Cardiac failure denoted by cyanosis, dyspnoea, and pulmonary edema, frothy to bloody cough, and the rapid thready pulse can best be treated by prompt administration of oxygen, digitalization, and intravenous administration of hypertonic glucose (50 cc. of a 50% sol.). If the right heart is dilated as is evidenced by the presence of dilated veins of the neck and face and by a marked cyanosis, 200 to 500 cc. of blood should be withdrawn by venesection and oxygen administered.

Hepatic and renal insufficiencies are the least understood and may be ushered in insidiously. Anorexia, vomiting, apathy, weakness, scanty urine at first and then a complete suppression (anuria) usually characterize this complication. Hiccoughs may be present just as in peritonitis and subdiaphragmatic abscess. Delirium and coma occur preterminally. Toxic effects of the anesthesia, surgical trauma, dehydration, and hypoproteinemia have all been listed as possible causes superimposed on an already damaged liver and kidney.

Important in the actual as well as in the prophylactic treatment of hepatic insufficiency is to keep the patient in good water balance (3000-5000 cc. per day) and to give plenty of glucose and protein. Glucose and protein when used together have been shown to have a far better protective action on the liver cells than when glucose is used alone. The protein inhibits deposition in the liver. Ravdin and Whipple have clearly shown that glucose and protein used together prevent hepatic necrosis.

Maintenance of adequate urinary secretion (adult 1500 cc. per day) denotes good kidney function. Should evidence of urinary suppression exist, prompt administration of 10% glucose sol. and physiological saline sol. given intravenously may produce diuresis. If this fails, plasma should be given. In the patient who has a severe sepsis or toxemia and whose urinary output is low, adrenal cortical extract in large doses is especially indicated. Finally as a word of caution one should remember that if saline solutions are used too zealously, a marked edema of the entire body may occur because of the salt retention due to the inability of the kidneys to excrete all of it. A good plan is to alternate the saline solution with 5% glucose solution and, if the protein level is below 7 mg. %, to give a transfusion of plasma.

* * * *

SALMONELLA

(Continued from page 18)

ican authors led by Edwards[3], Bornstein[2] and Mickle[4] decided upon the following policy:

1. An organism showing antigens that already have been accepted as Salmonella antigens and revealing typical morphological and biochemical properties of the genus is a Salmonella.

2. An organism showing a part of known Salmonella antigens and typical morphology and biochemical properties, or revealing typical known antigenic structure but having some slight deviation from the biochemical pattern of the genus may be accepted as a new member of the genus Salmonella, provided that it was isolated from an actual case of disease.

Besides the occurence of the type of deviation mentioned above, another eminent feature of Salmonella (and related genera) is the display of several types of bacterial variation. One of them is termed "loss variation," i.e. the loss of partial antigens, which leads to the formation of new types. It was upon this phenomenon that Andrewes based his theory that all Salmonella and related organisms stemmed from an original bacterium equipped with all partial antigens. On the other hand it is possible to induce new antigens by using the proper technique.

The "S"-"R" variation is common to all micoorganisms, but it plays an unusually important role in Salmonella. While the "S" form of the organism has good antigenic properties, being a satisfactory immunizing agent, represents the virulent form of the organism and displays typical biochemical features, the "R" form shows a partial or complete lack of these characteristics.

Another type of variation is the "V"-"W" variation, occurring in organisms revealing the "Vi" antigen. The term "V" applies to colonies of bacteria containing "Vi" antigen, while in the "W" forms

loss variation has taken place rendering the "Vi" antigen absent. The difference in the colony form may be readily seen, the "V" form having a different structure and greater opacity than the "W".

There are, however, additional differences between the "V" and "W" forms of Salmonella. Typhoid bacilli containing "Vi" antigen are more pathogenic for mice than organisms lacking this antigen. It has been well demonstrated, however, that high agglutination titers produced in animals by injections of "Vi" antigen does not imply protection against all the antigens of the typhoid bacillus, but it has been shown that animals injected with "V" forms demonstrate a higher protection against typhoid infection than those vaccinated with "W" forms. "V" organisms are more resistant to phagocytosis than "W", but the "V" colonies are readily attacked by bacteriophage. Because the phages are specific for the strain of typhoid upon which they act, this reaction is often used as a means of typing typhoid bacilli, thus permitting infections or outbreaks to be traced to their source.

As the "Vi" antigen disappears during the "V"-"W" variation, the "O" antigen alone then represents the somatic factors in agglutination experiments. It is important to note that in the serum of carriers the "Vi" antigen is agglutinated to a titer of 1:20 or higher. The results of agglutination experiments with "Vi" antigens in cases of disease are somewhat confusing, perhaps because of the variations in technique employed by different workers, but positive results can be obtained in most instances when proper methods are used.

As to the relationships between agglutination and animal protection they parallel one another to a certain degree, but actually have little in common. Luippold[7] proved that protection of the animal against all the components of the antigenic structure of a Salmonella does not protect that animal against that Salmonella organism itself. In our experiments, mice immunized against *S. typhi-murium* I. IV. XII: i-1. 2. 3 and *S. rubislaw* XI: r-enxz. did not protect against *S. aberdeen* XI: i-1. 2. 3. in spite of the presence of agglutinins against the partial antigen to a high titer in the blood.

While the phenomenon of cross-agglutination cannot be utilized for polyvalent immunization, it is successfully applied in the serological diagnosis of Salmonella fever. The best combination of antigens for use in the Widal reaction in the United States is the following series used by the New York Salmonella Center:

Alcoholized (somatic) antigens:

S. derby	IV
S. oranienburg	VI,VII
S. enteritidis	IX

Formolized (flagellar) antigens:

S. urbana	b
S. kentucky	i
S. muenchen	d
S. newport	1. 2. 3.

Ninety-seven per cent of the Salmonella types occurring in the United States can be revealed by agglutination of the patient's serum with the above antigens. Because of the rarity of *S. paratyphi* in this country its antigens have purposely been neglected here.

The Salmonella problem has been greatly clouded by the differences in the conception of their pathogenicity for man. The original attitude expressed by the German school, the so-called "Kiel doctrine," separated Salmonella organisms causing a typhoid-like fever in man from those of animal origin considered primarily pathogenic for animals and causing only occasionally a mild gastroenteritis in man. They supposed these two groups could be distinguished through a biochemical reaction, namely the fermentation of *d*-tartarate.

This idea has been largely replaced by American workers whose modern conception, named the "doctrine of Montevideo," regards all strains of Salmonella as potential pathogens for man and animals. It teaches, moreover, that the type of disease caused by Salmonella does not depend solely upon the "human" or "animal" adaption of the strain, but upon the resistance of the attacked individual. Children and young animals tend to be more inclined to septic invasion, meningitis, osteomyelitis, etc., than adults, as is demonstrated by the extensive statistics of Hormaeche[8], Edwards[9] and Seligman[9]. Too much importance is still given to a division of Salmonella into "paratyphoid bacilli" causing typhoid-like fevers, group "C" causing septicemia and the "enteritis" group causing "food poisoning". According to the observations of the above-mentioned investigators, as well as to the author's own experience, Salmonella infections should be classified as follows regardless of the type that causes the infection:

1. Salmonella fever, including "typhoid" and "paratyphoid" fever without localizing signs.
2. Salmonella septicemia (included by some authors in the first group) with localized processes due to hematogenous spread.

3. Predominant gastro-enteritis.
4. The carrier status.

Infection with Salmonella is acquired through the four "F"s: food, fingers, feces, flies. The reservoir of these infections are human carriers or animals, such as fowl, swine and rodents. The organisms are transferred to man by eating the meat of infected but apparently healthy animals, or by the contamination of food by human carriers, or with the excreta of sick or carrier rodents. Cherry et al[10] proved that a high per cent of the best quality inspected market meat contains Salmonella.

The human carrier represents a great menace, especially if he becomes a food handler. It is supposed that the carrier status is maintained by the invasion of the biliary system by Salmonella. The number of carriers among the general population is not known. We found about one per cent Salmonella and Shigella carriers in patients newly admitted to Illinois State Hospitals from the area of Greater Chicago[11].

In order to diagnose cases and to identify the carriers who present such a menace, blood and urine should be examined as well as feces. Modern laboratories use the plates of MacConkey, Liefson, Wilson-Blair and their modifications such as S.S. agar, Hajna-Perry etc. for Salmonella cultivation. Also recommended are the enrichment fluids of Liefson (Selenite) and of Mueller-Kauffman (Tetrathionate). Suspicious colonies are fished to Kligler's or Krumwiede's medium, then examined for biochemical activity.

Those organisms concurring with the specifications set up for Salmonella are sent to special laboratories for the diagnosis of the strain involved. Such laboratories carry all the necessary sera for typing and specially trained personnel are available. There are several Salmonella centers in the United States to which strains may be sent for identification, among which are P. R. Edwards, University of Kentucky, Agricultural Experiment Station or S. Bornstein, Division of Bacteriology, The Jewish Hospital of Brooklyn, Brooklyn, New York. While the identification of strains is not too important from the point of view of therapy, it is extremely important for epidemiological purposes and occasionally for prognosis.

Preventive vaccination against Salmonella in the United States is practiced against three strains, namely, *E. typhosa, S. paratyphi,* and *S. schottmuelleri.* Great Britain and the British colonies add *S. hirschfeldii* to this group. In cold countries only *E. typhosa* and *S. schottmuelleri* are used. The vaccination produces only an immunity specific for the type vaccinated against and not for other members of this group.

As to the treatment of Salmonella infections, soluble sulfa drugs (as sulfathiazole and sulfadiazine) are indicated in generalized infections, while sulfa drugs not readily absorbed from the intestinal tract (as sulfaguanidine and succinylsulfathiazole) are given in cases of gastroenteritis.

The bacteriophages, so excellent in the diagnosis of typhoid strains, did not prove to be valuable in therapy. The same thing is true of serum.

To summarize the practical implications of the present status of the Salmonella problem, the following chart demonstrates how various factors of Public Health work are set in action to control these infections:

	Veterinarian	Diagnostic Laboratory	Biological Laboratory	Epidemiologist	Public Health Engineer	Physician
Diagnosis of disease	(x)	x				x
Tracing source of infection to:						
food handlers		x		x		
food	x	x		(x)		
contamination during storage	x	x		x		
water		x		x	x	
General protection						
food inspection	x	x				
food storage	x	x		x	x	
food handllers		x		x		
animals	x	x	x			
Prophylactic immunization			x	x		x
Treatment of case	(x)		(x)			x
Carrier problem		x		x		x

Only the cooperation of all factors involved in this work can bring about the eradication of Salmonella infections. It is the intention of this paper, written for doctors and future physicians, to call their attention to the grave menace to health encompassed by the genus Salmonella. As the overlooking of a case of plague may lead to a permanent endemicity of this disease among rodents and through it may be a constant danger to man, so the overlooking, neglecting, or minimizing of "just a simple diarrhea" may lead to a carrier status with all the dangers embracing this circumstance. The patient may have "just a diarrhea," but his child can contract a fatal meningitis from him. It is hoped that the younger generation of modern physicians will have a broad and understanding view concerning the question of Salmonella infections.

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HYPERTENSION

(Continued from page 25)

anatomical lesions remain. After proper mounting and staining, the diameter of the lumen and the thickness of the arteriolar wall can be measured by means of a micrometric eyepiece. The measurements indicated in figure 3 are used to compute the wall to lumen ration which represents the thickness of the arteriolar wall in relation to the total size of the vessel. The value of the wall/lumen ration is low (0.3 to 0.7, ave. 0.5) in the normal individual in whom the arterioles have thin cells and large lumina

TABLE II

AVERAGE EFFECTIVE RENAL BLOOD FLOW, ETC., IN HYPERTENSIVE PATIENTS FIVE TO TWELVE MONTHS AFTER OPERATION

	Group I*		Group II		Total Average	
	Pre-op	Post-op	Pre-op	Post-op	Pre-op	Post-op
Effective Renal Blood Flow (cc/min.)	427.5	510.4	764.6	682.4	660.9	629.45
Filtration Rate (cc plasma/min.)	75.5	99.4	115.16	143.65	102.94	130.05
Filtration Fraction %	29.81	33.09	25.15	33.00	27.37	33.03
Blood Pressure on Admission	229/131	172/111	212/137	159/103	217/135	163/106
Blood Pressure During Clearance Test	197/128	169/110	176/113	159/102	183/117	160/104
Keith-Wagner Classification of Eyegrounds						
Normal	0	0	3	2	3	2
I	0	0	0	0	0	0
II	0	2	1	4	1	6
III	3	4	5	3	8	7
IV	3	0	0	0	3	0

*Pre-op averages computed on the data of only those patients who were reexamined.

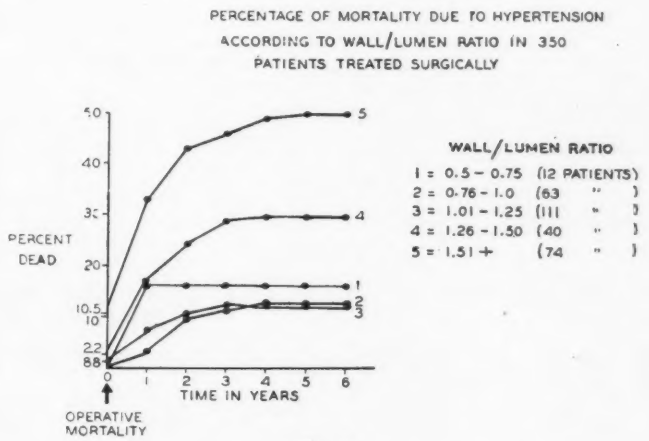

Fig. 6

and increases in hypertension up to 3.5 or more (Figure 4). The wall/lumen ratio was mesaured in muscle biopsies obtained from 350 consecutive cases of hypertension.[19] All cases were operated on for splanchnicectomy after the biopsy was taken and followed for 1 to 7 years after the operation. The results of this study indicate that there is a very significant correlation between thickening of the arterioles and the severity of the clinical symptoms, and that the prognosis is more severe in those cases in which the vascular lesions are more advanced (Figures 5 and 6).

SUMMARY

A method for the clinical study of arterial hypertension has been outlined. The tests can be divided in two categories; the first category includes the tests directed toward the study of the vascular system, such as the examination of the vasomotor reactions, of the eyegrounds, of the renal circulation and of the

arterioles in the skeletal muscles; the second category includes the tests of renal and cardiac function directed toward the study of the lesions which hypertension has produced in these systems.

It is believed that only a thorough and standardized method of examination of the patients will permit a careful evaluation of the numerous methods of therapy. The method suggested here has the advantage of having already been used, at least in part, in a large number of patients.[3]

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